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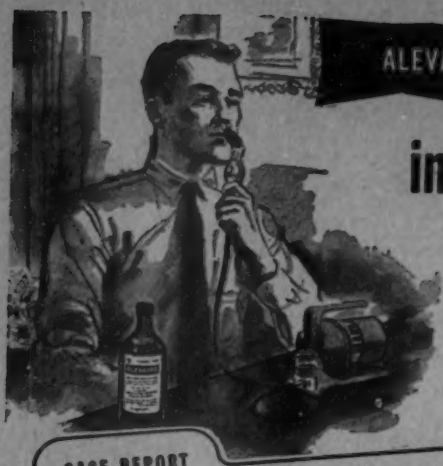

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*Miller, J.B., et al.: Ann. Allergy, 12:611, Sept.-Oct., 1954.

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THE
LARYNGOSCOPE.

VOL. LXVI AUGUST, 1956 No. 8

A NEUROTOLOGIC DIFFERENTIATION BETWEEN HIGH AND LOW LYING DISTURBANCES OF THE CENTRAL NERVOUS SYSTEM.*

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INTRODUCTION.

Prior to his death in 1934 Dr. E. R. Carpenter was the pioneer neurosurgeon of the Southwest. He had entered the field of neurosurgery after many years of active practice in ophthalmology and otolaryngology; consequently, he maintained an active interest in neurotology. Knowing of my primary interest in otology and the then young field of neurotology[1] I was privileged to see the majority of his cases that had neurotologic implications.

On March 24, 1931, we saw Mr. R. V. F., age 46, who complained of attacks of nonrotary vertigo with fulness of the ears but with no falling, staggering, emesis or sweating, beginning six months earlier. Placed on a restricted diet by his family physician he had improved. One week previously his attacks recurred, this time with "black-outs", and on two occasions total loss of consciousness with subsequent periods of mental confusion. During one attack his physician had observed rigidity of the left arm with extension and fanning of the left fingers. He was now having hallucinations of smell.

On examination his hearing was within normal limits, and his equilibratory apparatus revealed no abnormal spontaneous phenomena. On rotation, with his head 30° forward and then 120° forward, all responses were normal. The Barany caloric tests with water at 20° C. produced no response from either labyrinth. This was confirmed by the Kobrak test, using 10 cc. of water at 5° C. in each ear. Pneumoencephalograms and subsequent surgery revealed a large astrocytoma of the left temporal lobe.

* Submitted as Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1956.

Editor's Note: This ms. received in The Laringoscope Office and accepted for publication, January 20, 1956.

On November 16, 1933, we saw Mr. S. B., age 24. This patient complained of vertigo and staggering, of only one week's duration. He was growing worse rapidly. Hearing tests revealed slight right conduction deafness.

Examination of his equilibratory apparatus revealed spontaneous rotary nystagmus to the left of third degree. On left gaze it became severely oblique upward and left, and on upward gaze it became markedly vertical upward. On downward gaze it resumed its left rotary character. He past pointed six inches to the right with both hands. His gait was stiff and ataxic, there was a marked swaying Rombergism with his head in all the three positions; his pelvis was stiff, and his vertigo was disorganized.

On rotation to the right in both positions of the head his nystagmus was markedly exaggerated. It was of high amplitude and lasted 55 seconds. On rotation to the left his nystagmus was scarcely perceptible and lasted only 15 seconds. Both ears responded normally to the Barany caloric test with water at 20° C. A diagnosis of midline cerebellar tumor was confirmed at surgery.

Since several other cases seemed to support such a view I wondered whether these two cases had established a pattern of findings in high (probably supratentorial) and low (probably infratentorial) lesions.

By this time Dr. Carpenter's health was failing and he could no longer work with me on this problem. Other available neurosurgeons were uninterested in or antagonistic to the work, feeling that the otologist had little to offer, and that the rotation tests were actually dangerous to a patient with an intracranial lesion no matter how carefully they were done! Consequently my observations then became limited to the comparatively few and widely spaced cases that I encountered in my private otolaryngological practice. My interest did not flag; the problem was kept in mind, and whatever appeared in our journals concerning this condition was read and filed.

In 1951 I began making weekly trips to the United States Veterans' Administration Hospital at McKinney, Tex., in my capacity as consultant in otolaryngology to the Dallas and McKinney Hospitals. Since I was happy to see all cases indicating labyrinthine disturbance our department soon had as many such cases as we could handle.

Dr. Donald Corgill, the chief of service in otoloryngology at the hospital, became interested in these cases also. Since it is a field that receives but scant attention from so many clinicians, probably because of its time consuming nature, and so represented a new field of interest to them, our residents, most especially Drs. R. L. Duff and Jas. M. Anthony, became ac-

tively interested in this phase of otology. As these labyrinthine cases were worked out we discussed the anatomic and physiologic basis for our tests. At a general staff meeting, where our work in neurotology was being discussed, I mentioned my suspicion of the existence of high and low central nervous system lesion patterns and a possible anatomico-physiologic basis for such a pattern.

Dr. C. E. Kern, the chief of neurosurgery, also became interested. He is a neurosurgical student and a clinician of avid scientific interest, whose mind remains receptive to a new idea, even though suggested by an otologist. By his keen interest, his bitingly constructive criticism and his ready cooperation, we soon were able to see enough cases of suspected or already diagnosed intracranial disturbances (organic) to become of suggestive statistical significance.

It is on the basis of the original observation made 20 years ago, on the cases recently seen and reported here, and on reports in literature of scientific experimental work in neurology and neurotology by investigators of established ability and integrity, that this paper is written.

First, our method of examination will be described; then a report of a series of 52 consecutive cases seen in conjunction with the department of neurosurgery will be presented and analysed, and finally the anatomico-physiologic problem will be presented and developed to present a concept of the possible structures and pathways involved.

METHOD OF EXAMINATION.

A careful history is taken of all cases showing dysfunction of the equilibratory system. The patient is allowed to tell his own story, and then any pertinent questions are asked. This is followed by a physical examination of the ears, nose and throat.

A complete audiological examination, including high and low tone thresholds, the pure tone threshold curves by both air and bone conduction, speech reception ability and the presence or absence of recruitment and diplacusis is the first step of the actual examination of the cochleo-vestibular apparatus.

This apparatus we hold to be a remote but integral part of the central nervous system, its upper limit being the cerebral cortex.

The examination of the canalo-vestibular portion of the cochleo-vestibular organ is begun by having the patient attempt to walk a straight line, with his eyes closed, to touch a point 20 feet ahead of him, observing his gait for steadiness, staggering or deviation.

We then observe his Romberg response with his face forward, to the right and to the left; his pelvic girdle reaction to sudden tilt to right, to left, backward and forward; the presence or absence of nystagmus with the head upright gazing straight ahead, to the right and to the left, then with the head tipped backward to a horizontal position while gazing upward to the ceiling, then with the head tipped laterally to the right shoulder and finally tipped laterally to the left shoulder while gazing on a level line. The presence or absence of past pointing is then established. His ability to detect rotary motion and its after-response is then observed by placing him in the turning chair and, with his eyes closed, turning him once slowly to the right and then after a slight pause, once slowly to the left.

The results of this portion of the examination we designate the spontaneous phenomena.

The classical Barany turning tests are then performed to determine at least nystagmus and post-rotary vertigo with the head tipped 30° forward. If any response calls for further turning, the head then is brought forward to the 120° position; but here the rotation is at the speed of 10 turns in 20 seconds, or one-half the speed with the head in the 30° position. This change is made because the physical effect of centrifugal force is so much enhanced by increasing the distance, equal to the upper body length, from the center of rotation.

I consider the turning tests to be a *sine qua non* of the examination of the equilibratory apparatus. The normal function of this apparatus is to respond to position and to motion. The turning tests probe this function; therefore they and not the caloric tests are the true physiologic tests, and failure to perform them must be accounted negligence.

The caloric tests, next in the order of the examination, formerly (prior to 1940) were carried out according to the Barany technique, using a continuous irrigation at 20° C. with the head 30° forward, and timing from the beginning of the irrigation to the onset of the nystagmus. Forty-five seconds was held to be normal. Decrease or increase of this time was held to indicate hyperactivity or hypoactivity; but the accuracy of these tests was subject to valid objections under certain conditions.

Because of its ease and accuracy I have changed to the Atkinson technique,[2] and in all cases since 1940 I have used this method as the basic caloric test in the following manner:

A small pan is filled with chipped ice and a small quantity of tap water is added. Within a matter of moments this system of melting ice attains a temperature of 0° C. This temperature will maintain itself if the pan is kept covered until the ice has almost completely melted. A 1 cc. tuberculin syringe (barrel and plunger separated) is placed in this solution and allowed to remain for about five minutes. Due to the poor heat conducting quality of glass and to the relatively thick wall of the syringe in relation to the small volume of water in the barrel the temperature of the water in the filled syringe will not change in the very few seconds required to convey it from the pan to the patient's ear.

The patient is tipped to the left until the head is in the left lateral horizontal position and then the chin is rotated upward about 30°, which brings the horizontal canal into position for a maximum response, as it is the horizontal and not the vertical canal which responds to this test producing the initial rotary vertigo. The patient then is instructed to gaze at a point straight ahead and is warned not to move when the cold water is instilled into the ear. The chilled syringe is filled with 1 cc. of the water at 0° C. and promptly discharged into the external auditory canal. A split second two-handed stop watch is started at the same instant, and the eyes of the patient are watched intently for the first evidence of nystagmus. As experience is required to make this observation with accuracy the resident is required to do this with me using his own stop watch. After a surprisingly few number of observations we

rarely will differ by more than a fraction of a second in spotting the first twitch. As soon as this first twitch occurs the first of the two hands of the stop watch is stopped to signal the end of the latent period. As soon as we are assured that the nystagmus has started and its direction has been noted the patient is brought to the upright (head 30° forward) position allowing the water to drain from the ear, the eyes are closed, and the presence or absence of past pointing is established. This tests the reactions commonly ascribed to the vertical canals. The head is then tipped backward to a horizontal position, the eyes are opened, and the patient is instructed to look at the ceiling. The direction and the degree of the nystagmus are observed again. The eyes are closed and the past pointing reaction is observed in this position. This tests the reaction commonly ascribed to the horizontal canal. The head is returned to the upright position, the eyes are opened and fixed on a point straight ahead and the nystagmus is watched until it ceases. At this instant the second of the two hands of the stop watch is stopped. The time thus recorded, minus the latent period, represents the duration of the nystagmus.

In normal cases the ratio of the latent period to the duration is established quite constantly as 1 : 4. This ratio we speak of as the Atkinson ratio.[2] It is the ratio and not the time that is important. The average normal latent period we have found to be about 20 seconds, and the duration time to be about 80 seconds or 1 : 4; but a latent period of 15 seconds and a duration time of 60 seconds still would be normal, as would a latent period of 25 seconds and a duration time of 100 seconds.

If the ratio is altered it indicates altered action of the equilibratory apparatus. Shortening of the latent period in relation to the duration time, *e.g.*, 15 seconds to 75 seconds, or 1 : 5, denotes hyperactivity or irritation. Lengthening of the latent period in relation to the duration time, *e.g.*, 25 seconds to 75 seconds, or 1 : 3, indicates hypoactivity by suppression or blocking.

The patient is allowed to rest for about 15 minutes, or at least until all evidence of reaction to the test has subsided. The left ear then is tested in the same manner. All caloric tests in the cases herein reported were performed according to this

technique. So accurate and so easily mastered is this test that at least three correct localizations in these cases were made by the resident staff in cases that required an immediate report at a time that I was unavailable.

The results obtained by the rotational and the caloric tests we designate as the induced phenomena.

REPORT AND ANALYSIS OF CASES.

With the exception of the two cases mentioned in the Introduction, which are not included here, and the last case to be reported which was seen by me fortunately, as well as fortuitously, in our private practice just as the series was closed, all the cases were at the U. S. V. A. Hospital at McKinney, Tex. All are white males. They are all the cases seen in continuous chronological series (correctly and incorrectly localized or undiagnosed) in conjunction with the department of neurosurgery; therefore they are unselected and are controlled as to diagnosis.

The cases are numbered in the chronological order in which we saw them, but for convenience in presentation they have been regrouped as to localization where localization was correct, and as to incorrect and unconfirmed localizations. The case numbers as presented in the report do not appear in the order in which they were seen, but they are all present—from Case No. 1, seen August 19, 1951, to case No. 52, seen privately, June 18, 1955.

Cases confirmed by neurosurgical, neurological or medical diagnosis with their localization are as follows:

Those numbered 6, 7, 9, 18, 28, 33, 34, 45, 49 and 52 are high, localized lesions.

Nos. 2, 35, 10, 1, 16, 25, 26, 31, 50 and 44 are high, diffuse lesions.

Nos. 5, 40 and 30 are low lesions, localized and diffuse.

Nos. 37, 46 and 38 are low, localized lesions with high extension.

Nos. 3, 4, 14, 19, 29, 32, 43, 47 and 27 are lesions located near the tentorium, with either high or low extension.

Nos. 11, 12, 17, 22, 24, 39 and 42 are diffuse lesions with widespread generalized high and low symptoms.

Definite errors in diagnosis or cases that were temporarily unconfirmed by neurosurgical or neurological opinion are numbered 8, 13, 15, 21, 23, 36 and 41. Those numbered 20, 28 and 51 are lesions of the cerebello-pontine angle.

CASE No. 6: J. B. F., age 39. Admitted March 5, 1952. Examined March 14, 1952. Reg. No. 34108.

History: In October, 1949, this patient suffered a severe headache, rotary vertigo and pain and limp in the left leg. Diagnosed at Brook General Hospital as "cyst of the third ventricle." Nothing was done at this time. On May 21, 1951, he entered this hospital and the case diagnosed as supracellar cyst or craniopharyngecma; inoperable. The patient left this hospital and a private neurosurgeon did a Thorkildsen procedure. This was followed by severe aseptic meningo-encephalitis. On March 5, 1952, he returned to this hospital with residual symptoms indicating "bilateral pyramidal and extra-pyramidal disease indicating affection of both basal ganglia and internal capsules, perhaps also involving the hypothalamus."

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 22 secs.	Hor. to right. *Amp.*—Good; Dur. 22 secs.
Vert.—Good; no autonomic phenomena.	Good; no autonomic phenomena.

HEAD 30° FORWARD.

D. *Caloric*—Right ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 55 secs. Dur. 50 secs.	Rot. to right. *Amp.*—Good; After 35 secs.; Dur. 62 secs.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Atkinson Ratio—1:1-;	1:2-,

Summary—Response to rotation is normal; response to caloric test markedly subnormal.

Neurosurgical Diagnosis—Supracellar cyst.
Meningo-encephalitis with residual pyramidal and extrapyramidal tract involvement.

CASE No. 7: L. G., age 30. Examined March 20, 1952. Reg. No. 34566.

History: This patient was operated upon in this hospital on September 18, 1951. At that time a deep lying astrocytoma was found in the right

Fig. 1. Encephalogram of Case No. 6 showing marked dilation of the lateral ventricles from blocking in the third ventricle.

temporal lobe. He was sent to the department of otolaryngology without history for our evaluation.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 27 secs.	Hor. to right. *Amp.*—Good; Dur. 27 secs.
Vert.—Good; no autonomic phenomena.	Good; sweat and pallor.

HEAD 120° FORWARD

Nystagmus—Rot. to left. *Amp.*—Good; Dur. 17 secs.	Rot. to right. *Amp.*—Good; Dur. 15 secs.
Falling—To right; good.	To left; good.
Vert.—Good; pallor and sweat.	Good; pallor and sweat.

HEAD 30° FORWARD

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—None.	Rot. to right. *Amp.*—Good after 26 secs.; Dur. 92 secs.
Vert.—Subnormal; pallor and sweat.	Subnormal pallor and sweat.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—Subnormal.	Subnormal.
Atkinson Ratio—Not Obtainable.	1:3.5.

Summary—Response to rotation normal; right vertical tracts completely blocked; left vertical tracts subnormal to caloric test.

CASE No. 9: W. L. H., age 30. Admitted April 14, 1952. Examined April 18, 1952. Reg. No. 34947.

History: Two and one-half weeks ago upon straightening up after a backward bent position while at work, patient suffered a severe attack of pain and numbness in the left occiput, neck, shoulder, arm and left chest. The left hand failed to grasp objects properly; *i.e.* he did not place his hand in accordance with the shape of an object (thalamic symptom?).

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 24 secs.	Hor. to right. *Amp.*—Good; Dur. 28 secs.
Vert.—Good; slight pallor and sweat.	Good; slight pallor and sweat.

HEAD 120° FORWARD.

Nystagmus—Rot. to left. *Amp.*—Good; Dur. 15 secs.	Rot. to right. *Amp.*—Good; Dur. 15 secs.
Falling—Good to right.	Good to left.
Vert.—Good; pallor, sweat and nausea.	Good; pallor, sweat and nausea.

HEAD 30° FORWARD.

D. *Caloric*—Right ear.	Left ear.
Nystagmus—Rot. to left. *Amp.*—Fair; After 35 secs.; Dur. 90 secs.	Rot. to right. *Amp.*—Fair; After 37 secs.; Dur. 78 secs.
Vert.—Marked; pallor, sweat and emesis.	Good; slight nausea.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Fair.	Hor. to right. *Amp.*—Fair.
Vert.—Good.	Good.
Atkinson Ratio—1:2.6.	1:2.

Summary—Response to rotation normal; response to caloric test markedly subnormal especially on the left.

Neurotologic Diagnosis—A high central lesion, probably in the region of the thalamus.

Neurosurgical Diagnosis—A vascular accident in the upper brain stem (mesencephalic) region up to the lower thalamic nuclei.

CASE No. 18: W. E. A., age 32. Admitted November 20, 1952. Examined November 21, 1952. Reg. No. 39370.

History: Pain in the right ear and mastoid and in the left frontal area for one month. Since then increasing diplopia, nausea and staggering.

EXAMINATION.

A. *Hearing*—A moderate bilateral *conductive* type impairment.

B. *Spontaneous Phenomena*—Normal except a deviation of 5 feet to the left in walking the 20 feet line.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. Amp.—Good; Dur. 25 secs.	Hor. to right. *Amp.*—Good; Dur. 27 secs.
Vert.—Fair; little or no autonomic phenomena.	Fair; moderate autonomic phenomena.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left ear.
Nystagmus—Rot. to left. *Amp.* Poor; After 15 secs.; Dur. 85 secs.	Rot. to right. *Amp.*—Small; After 30 secs.; Dur. 88 secs.
Vert.—Good; nausea, pallor and sweat.	Fair; subnormal autonomic phenomena.

Fig. 2. Case No. 28. Combined encephalogram and arteriogram showing the space occupying mass in the parasagital region of the left temporal lobe.

HEAD BACK.

Nystagmus—Hor. to left. Fair to good.	*Amp.*	Hor. to right.	*Amp.*—Fair to good.
Vert.—Good.		Good.	
Atkinson Ratio—1:5.6.		1:3-.	

Summary—Response to rotation normal; response to caloric test showed right tracts markedly hyperactive and left tracts equally hypoactive.

Neurotologic Diagnosis—High lesion on left.

Neurosurgical Diagnosis—Operation on November 24, 1952. Partial removal of glioblastoma mutiforme from left temporal lobe extending to basal ganglia.

CASE No. 28: H. D. A., age 32. Admitted September 25, 1953. Examined November 13, 1953. Reg. No. 45492.

History: Onset April 4, 1953, with a severe convulsion followed by unconsciousness. A short time later there was another convulsion. At another institution the patient was placed on dilantin for two months, during which there was apparent improvement with only a few episodes of unconsciousness. The convulsions then resumed and there was clumsiness of the right leg and slurring of speech. Arteriograms and encephalograms revealed a space occupying lesion in the para-sagittal region of the left temporal lobe. At surgery on October 12, 1953, a glioma in that area was found. The patient was sent to us with a known localized lesion against which we could calibrate our tests.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.* Good; Dur. 22 secs.	Hor. to right. *Amp.*—Good; Dur. 26 secs.
Vert.—Subnormal.	Subnormal.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.* Good; After 22 secs.; Dur. 122 secs.	Rot. to right. *Amp.*—Good; After 22 secs.; Dur. 115 secs.
Vert.—Subnormal to fair.	Fair to good.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Large.	Hor. to right. *Amp.*—Large.
Vert.—Subnormal to fair.	Fair to good.
Atkinson Ratio—1:5.5.	1:5.2.

Summary—Response to rotation normal; response to caloric test showed hyperactivity.

Neurotologic Diagnosis—A postoperative high central lesion.

CASE No. 33: J. K. H., age 29. Admitted April 11, 1954. Examined April 30, 1954. Reg. No. 49236.

History: All his life this patient had a marked inability to meet life's ordinary stresses, but he had a marked libido with strong sex powers. About nine months ago he began to get into financial troubles. He developed headaches, became more indecisive, his sex powers lessened and became perverted, there was increasing somnolence with disorientation, urinary incontinence and a marked increase of water intake.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

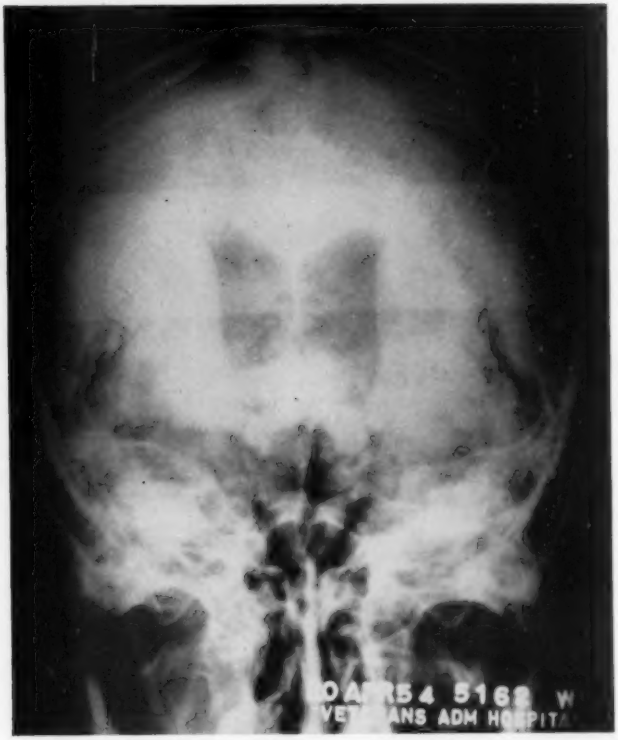

Fig. 3. Case No. 33. Anterior-posterior encephalogram showing dilated ventricles with tumor of the third ventricle.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 30 secs.	Hor. to right. *Amp.*—Good; Dur. 28 secs.
Vert.—Good; marked pallor and sweat.	Good; marked pallor and sweat.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Fair after 43 secs.; Dur. 102 secs.	Rot. to right. *Amp.*—Poor after 47 secs.; Dur. 104 secs.
Vert.—Little or none; no autonomic phenomena.	Little or none; no autonomic phenomena.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.* Fair.	Hor. to right. *Amp.*—Poor.
Vert.—Little or none.	Little or none.
Atkinson Ratio—1:2.4.	1:2.2.

Neurotologic Diagnosis—Lesion high—probably floor third ventricle near hypothalamus.

Encephalogram—Dilated lateral ventricles with tumor anterior portion third ventricle.

Neurosurgical—On May 10, 1954, a massive tumor (malignant adamantinoma) filling the third ventricle.

Comment—This tumor was beginning to exert pressure on the posterior fossa structures.

CASE No. 34: R. S. J., age 31. Admitted September 27, 1954. Examined October 15, 1954. Reg. No. 52679.

History: This patient complained of nervousness, indecisiveness, headaches (much worse during the past five years), parosmia, and increasing inability to think. There had been one recent attack of loss of consciousness.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—A first degree nystagmus, greater on looking to the right than to the left. Otherwise within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.* Good; Dur. 25 secs.	Hor. to right. *Amp.*—Good; Dur. 23 secs.
Vert.—Good; slight pallor and sweat.	Good; no autonomic phenomena.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Small after 31 secs.; Dur. 84 secs.	Rot. to right. *Amp.*—Small after 34 secs.; Dur. 83 secs.

HEAD BACK.

Vert.—Fair.	Fair.
Atkinson Ratio—1:2.7.	1:2.5.

Summary—Responses to rotation normal. Responses to caloric tests markedly depressed.

Neurotologic Diagnosis—A high central lesion.

Encephalogram and Arteriogram—A tumor in the right frontal region—Left hydrocephalus.

Neurosurgical—On October 18, 1954, a large astrocytoma was found at operation in the infero-medial half of the right frontal lobe.

CASE No. 45: S. D. M., age 26. Admitted March 25, 1955. Examined April 15, 1955. Reg. No. 56368.

History: On October 10, 1954, this man suffered with a transient roaring throughout his entire head. Since that time he has had nine such

attacks, each accompanied with dimness of vision and followed by numbness of the left arm and leg. Otherwise his history is completely negative.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 28 secs.	Hor. to right. *Amp.*—Good; Dur. 27 secs.
Vert.—Good; no autonomic phenomena.	Good; no autonomic phenomena.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Fair after 52 secs.; Dur. 83 secs.	Rot. to left. *Amp.*—Fair after 16 secs.; Dur. 103 secs.
Vert.—Good; sweat.	Good; sweat, pallor and nausea.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to left. *Amp.*—Good.
Vert.—Good.	Good; sweat, pallor and nausea.
Atkinson Ratio—1:1.6.	1:6.4.

Summary—Response to rotation normal. Responses to caloric test show right markedly depressed; left markedly irritated.

Neurotologic Diagnosis—High blocking (primary) lesion on right; high irritation left.

Encephalograms—Ventricular dilatation and tumor in the right frontal lobe.

Neurosurgical Diagnosis—Operation on April 18, 1955, revealed a glioma in the posteroinferior portion of the right frontal lobe.

CASE No. 49: H. T. C., age 49. Admitted May 25, 1955. Examined June 1, 1955. Reg. No. 57716.

History: This normally loquacious right-handed individual was entirely normal until three weeks ago, when he became morose and disinclined to talk. One week ago he became unable to speak intelligibly, uttering only mumbling sounds. There was some vomiting of a profuse mucoid material.

EXAMINATION.

There was hemiparesis generalized on the right including all branches of the facial nerve; also a marked nominal aphasia although he understood perfectly, carrying out all directions as we performed the tests on his ears.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—Except deviation to the right of six feet (a result of this hemiparesis?) all were within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 29 secs.	Hor. to right. *Amp.*—Good; Dur. 27 secs.
Vert.—Good; nausea and retching.	Good; sweat and emesis.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good after 20 secs.; Dur. 107 secs.	None.
Vert.—Marked; emesis, pallor and sweat.	Marked; emesis, pallor and sweat.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor to right. *Amp.*—Good.
Vert.—Marked.	Good.
Atkinson Ratio—1:5.3.	Not obtainable.

Neurotologic Diagnosis—High lesion on left involving Broca's area.

Summary—All responses normal except from vertical tracts on left to caloric test.

X-ray of Chest—Inoperable carcinoma (bronchogenic) left upper pulmonary lobe.

Final Diagnosis—Metastasis bronchogenic carcinoma to Broca's area left frontal lobe.

Patient expired in this hospital July 12, 1955. Autopsy revealed metastatic tumor in left temporal lobe.

CASE No. 52: G. B. D., age 41. Private practice. Seen and examined June 18, 1955.

History: This man was operated on in 1925 by Dr. Carpenter, who removed a suprasellar cyst. The approach was right temporal area. Since surgery he has been totally blind and partially paralyzed in the left arm and leg. His hearing so far as he knew was normal. About two years ago he awakened one morning to find himself totally deaf in the left ear. He does not recall any unusual dizziness at that time.

EXAMINATION.

A. *Hearing*—Right ear normal; left ear total deafness.

B. *Spontaneous Phenomena*—Nystagmus first degree on right and left lateral gaze. Vertical on upward gaze. Otherwise normal.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Poor; Dur. 15 secs.	Hor. to right. *Amp.*—Poor; Dur. 15 secs.
Vert.—Poor.	Poor.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Obl. to left. *Amp.*—Huge after 15 secs.; Dur. 130 secs.	None.
Vert.—Good; sweat.	Questionable; no autonomic phenomena.

HEAD BACK.

Nystagmus—Obl. to left. *Amp.*—Huge irregular.	None.
Vert.—Good.	None.
Atkinson Ratio—1:8.6.	

Summary—On rotation the canals respond as with one labyrinth dead and compensated; on caloric test the right ear shows marked hyperactivity—central irritation.

Diagnosis—Postoperative high central irritation; total occlusion left cochleo-vestibular artery.

CASE No. 2: J. P. H., age 59. Admitted November 3, 1951. Examined November 30, 1951. Reg. No. 31728.

History: For the past six years this patient had suffered a persistent and progressive sense of imbalance and vertigo with staggering.

EXAMINATION.

A. *Hearing*—Within normal limits for age.

B. *Spontaneous Phenomena*—A tendency to backward sway in all three positions of the head, and a spastic gait; otherwise normal.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 25 secs.	Hor. to right. *Amp.*—Good; Dur. 23 secs.
Vert.—Good; no autonomic phenomena.	Good; slight nausea.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—None.	Rot. to right. *Amp.*—Small after 27 secs.; Dur. 92 secs.
Vert.—None.	Poor.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Very small.
Vert.—Little or none.	Little or none.
Atkinson Ratio—.....	1:3.4.

Summary—Responses to rotation are normal; responses to caloric testing show none from the right vertical tracts, subnormal from the left vertical tracts, normal from the right horizontal tracts and markedly subnormal from the left horizontal tracts.

Neurotologic Diagnosis—A diffuse process, probably vascular, high in the brain stem.

Neurologic Diagnosis—Diffuse cerebral arteriosclerosis.

CASE No. 35: H. H., age 40. Admitted October 8, 1954. Examined October 22, 1954. Reg. No. 52941.

History: This patient complained of "numbness" of the left side of his face. Three weeks previously he suddenly fell to the ground, his neck became stiff, and he quickly lapsed into unconsciousness. He remained unconscious for 24 hours, and was left with the numbness and weakness of the left side of the face, and with a limp of the left leg. Spinal tap at the time of the attack revealed blood in the fluid.

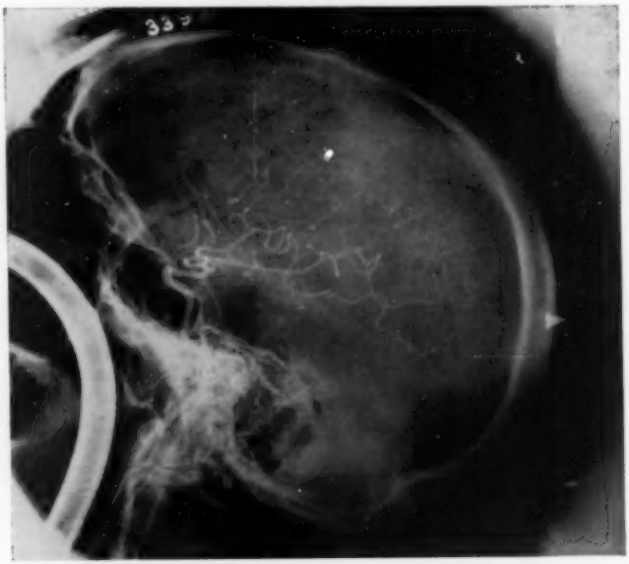

Fig. 4. Case No. 35. Right carotid arteriogram with no conclusive demonstration of a normal anterior cerebral artery.

EXAMINATION.

There was a subtotal paralysis of the left facial nerve typical of a supranuclear lesion.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.
Nystagmus—Hor. to left. *Amp.*—Large; Dur. 28 secs.
Vert.—Good; profuse sweat; severe emotional reaction—crying.

To left.
Hor. to right. *Amp.*—Large; Dur. 25 secs.
Good; profuse sweat; less emotional reaction.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.		Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good after 37 secs.; Dur. 99 secs.		Rot. to right. *Amp.*—Fair after 43 secs.; Dur. 72 secs.
Vert.—Fair.		Fair.

HEAD BACK.

Nystagmus—Rot. to left. *Amp.*—Good.	Rot. to right. *Amp.*—Fair.
Vert.—Fair.	Fair.
Atkinson Ratio—1:3.	1:1.6.

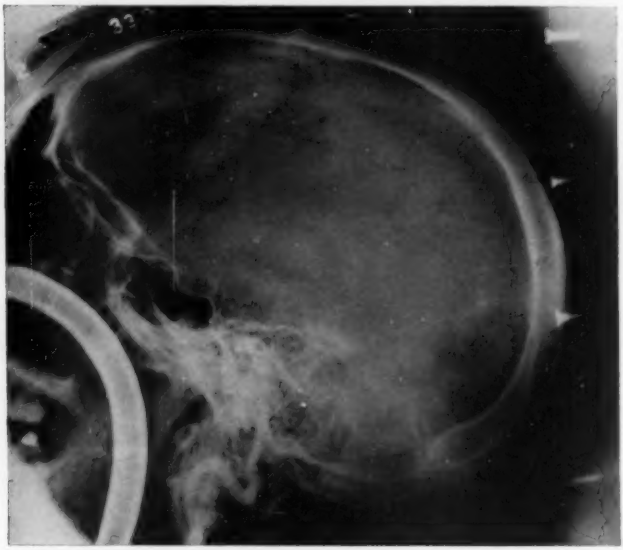

Fig. 5. Case No. 35. Right vertebral arteriogram showing only the vertebral and the beginning of a basal artery

Summary—Responses from rotation are normal; responses from caloric tests are subnormal on the right and markedly subnormal on the left.

Neurotologic Diagnosis—A high stem lesion on the left.

Arteriograms—These show *1.* A relatively small right carotid artery with no conclusive demonstration of a normal anterior cerebral artery on the right side (the vascular system is atrophic or anomalous). *2.* The left vertebral artery angiogram shows only the vertebral and the beginning of a basilar artery.

Neurosurgical Diagnosis—A subarachnoid hemorrhage in the region of the basilar artery.

CASE No. 10: W. O. S., age 52. Admitted May 5, 1952. Examined May 9, 1952. Reg. No. 35373.

History: This patient has had a suboccipital headache, persistent for ten or twelve years. Since 1950 he has had episodic severe pain in the left eye. He says the hearing in the left ear is impaired.

EXAMINATION.

The left pupil is smaller than the right. Weakness of the left palate and uvula with diminished gag reflex on the left. Diminished corneal reflex on the left and diminished taste on the anterior two-thirds of the tongue on the left. Diminished sense of touch in all branches of the fifth nerve. There is weakness of the left hand. The blood pressure is 250/150.

A. *Hearing*—The right ear is normal for his age; the left ear shows moderate downward curve loss in the upper registers.

B. *Spontaneous Phenomena*—His gait was ataxic and he tended to fall to the left in the Romberg test in all positions of the head; otherwise within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 27 secs.	Hor. to right. *Amp.*—Good; Dur. 32 secs.
Vert.—Good; slight nausea and sweat.	Good; no autonomic phenomena.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—good; After 43 secs.; Dur. 96 secs.	Rot. to right. *Amp.*—Good; After 30 secs.; Dur. 99 secs.
Vert.—Fair; nausea.	Good; nausea, emesis, profuse sweat.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—Fair.	Good.
Atkinson Ratio—1:2.2.	1:3.3.

Summary—Responses via the tracts from the right ear to rotation are exaggerated; responses to the caloric test are depressed, markedly so from the right ear.

Neurotologic Diagnosis: A high diffuse lesion most marked on the right side.

Neurosurgical Diagnosis: An undiagnosed intracranial lesion as the patient refused to permit an arteriogram or encephalogram.

In April, 1955, he returned to the neurosurgical service for re-evaluation. At that time the diagnosis was degenerative central nervous system disease probably syringomyelia.

On May 17, 1955, he committed suicide by shooting himself in the head because of unbearable headache.

CASE No. 1: A. G. G., age 40. Admitted August 13, 1951. Examined August 17, 1951. Reg. No. 30027.

History: This patient complained of pain on the right side of the body (typical Sluder's syndrome to ear, neck, shoulder, arm and leg), inability to have an erection, although he achieves orgasm without erection; depression, "don't give a damn"; contemplating suicide, and illusions of persecution involving, especially, his wife. Since childhood he states that he passes out easily but without convulsions. In 1931 he was injured in an auto wreck, being unconscious for two hours. In 1941 he was struck on the chin with an eight-pound sledge hammer and was unconscious for one hour.

EXAMINATION.

Negative except decreased sensation to pin prick on right forehead and scalp and on the left shoulder and arm.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 24 secs.	Hor. to right. *Amp.*—Good; Dur. 22 secs.
Vert.—Good; no autonomic phenomena.	Good; pallor, sweat and nausea.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—None.	None.
Vert.—Fair; pallor, sweat and nausea.	Fair (delayed); subnormal autonomic phenomena.
Atkinson Ratio—.....	.....

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—Good.	Fair.

Summary—Responses to rotation normal; responses from vertical tracts bilateral absent from caloric stimulation.

Neurotologic Diagnosis—A central cortical or high stem lesion.

Neurosurgical Diagnosis—A predisposition, severe, with a marked obsessive-compulsive character developed in a reaction to hostile mother fantasies and defects of emotional control and conceited intellectual performances. All probably residual to a childhood influenzal encephalitis or to severe head injuries during adolescence.

CASE No. 16: H. M. S., age 22. Admitted October 21, 1952. Examined October 31, 1952. Reg. No. 38770.

History: Twelve hours previously there was a sudden severe headache, bifrontal and radiating to both temples with hallucination of vision in the left eye. This was followed quickly by bilateral blindness, mania and unconsciousness.

EXAMINATION.

A. *Hearing*—The audiogram showed a bilateral drop to 35 db at 4,000 cps. Otherwise within normal limits.

B. *Spontaneous Phenomena*—A large horizontal nystagmus to the left, especially on slight *right* lateral gaze. On far right lateral gaze this changed to first degree to the right. In the back horizontal position of the head there was a rotary nystagmus to the left. Otherwise all within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 30 secs.	Hor. and Rot. to right. *Amp.*—Good; Dur. 43 secs.
Vert.—Subnormal; no autonomic phenomena.	Subnormal; no autonomic phenomena.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 18 secs.; Dur. 102 secs.	Rot. to right. *Amp.*—Huge after 22 secs.; Dur. 158 secs.
Vert.—Little or none.	Little or none.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—Little or none.	Little or none.
Atkinson Ratio—1:5.6.	1:7.2.

Summary—Responses to rotation show moderate hyperactivity especially from the right tracts; caloric test shows marked hyperactivity especially from the left tracts.

Neurotologic Diagnosis—Cerebral or high stem irritation with left cerebellar involvement.

Neurosurgical Diagnosis—Acute brain syndrome (cerebral vascular occlusion).

CASE No. 25: M. D. R., age 42. Admitted September 20, 1953. Examined October 30, 1953. Reg. No. 45365.

History: This patient complained of attacks of severe headache with loss of consciousness noted especially just as he would be falling asleep. There is a moderate diplopia which caused a recent car wreck. Condition progressive over several years.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—Deviated 3 feet to the left. Otherwise all within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 21 secs.	Hor. to right. *Amp.*—Good; Dur. 23 secs.
Vert.—Fair; no autonomic phenomena.	Fair to good; no autonomic phenomena.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 22 secs.	Rot. to right. *Amp.*—Fair after 35 secs.
Vert.—Fair to good.	Poor to fair.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Fair.	Hor. to right. *Amp.*—Good.
Vert.—Poor.	Good.
Atkinson Ratio—1:5.2.	1:2.8.

Summary—Responses to rotations are normal; to the caloric test, hyperactivity on the right and marked hypoactivity on the left.

Neurotologic Diagnosis—A high central lesion, nature unknown.

Encephalogram—Cerebral atrophy.

Neurosurgical Diagnosis—Post-traumatic cerebral atrophy with traumatic epilepsy.

CASE No. 26: L. R. G., age 35. Admitted October 24, 1953. Examined November 6, 1953. Reg. No. 46057.

History: In September this soldier suffered a shell injury to the left fronto-temporal area. In February, 1945, a metal plate was set at this site. Since that time he has had persistent dull aching bilateral frontal headache, partially relieved by aspirin. There were also occasionally mild episodes of dizziness with vomiting. Two weeks ago there was an episode of severe headache with dizziness and sub-total loss of consciousness.

EXAMINATION.

A scar in the left fronto-parietal area where X-ray shows a metal plate. The eye grounds show a yellowish area with a small amount of pigmentation, in the bilateral superior temporal areas. There was a moderate emotional flattening with memory deficit.

A. *Hearing*—Normal except that the audiogram showed a bilateral nicking at 5792 cps. on the right to 25 db and on the left to 60 db.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 24 secs.	Hor. to right. *Amp.*—Good; Dur. 23 secs.
Vert.—Good; sweat and nausea.	Good; pallor, sweat and nausea.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—None.	Rot. to right. *Amp.*—Large; after 42 secs.; Dur. 105 secs.
Vert.—Fair.	Good; nausea and emesis.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Large.	Hor. to right. *Amp.*—Huge.
Vert.—Marked. Sweat, nausea and emesis.	Marked. Sweat, nausea and emesis.
Atkinson Ratio—.....	1:2.5.

Summary—Responses to rotation are normal; caloric test shows absence of response from right vertical tracts, hypoactivity from the left vertical tracts, and marked hyperactivity from both horizontal tracts.

Neurotologic Diagnosis—Posttraumatic high central lesion.

Neurosurgical Diagnosis—Chronic brain syndrome; posttraumatic.

CASE No. 31: H. B., age 28. Examined Jan. 26, 1954. Reg. No. 47563.

History: The hospital record on this case has been lost. We have only the note on the consultation request from neurosurgery that it is a central lesion, and we have on our record the final diagnosis after our examination from neurosurgery.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 31 secs.	Hor. to right. *Amp.*—Good; Dur. 29 secs.
Vert.—Sub-normal.	Sub-normal.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Fair; after 40 secs.; Dur. 80 secs.	Rot. to right. *Amp.*—Fair; after 40 secs.; Dur. 85 secs.
Vert.—Sub-normal.	Subnormal.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—Sub-normal.	Subnormal.
Atkinson Ratio—1:2.	1:2.1.

Summary—Responses to rotation slightly exaggerated especially from left tracts; responses to the caloric test are markedly depressed.

Neurotologic Diagnosis—A high generalized lesion with early extension downward possible.

Neurosurgical Diagnosis—Chronic brain syndrome.

CASE No. 50: R. S., age 30. Admitted May 10, 1955. Examined June 17, 1955. Reg. No. 57290.

History: This right-handed patient was rendered unconscious from a blow with a blunt weapon on the right parietal area on this date. He was unconscious on admission, and X-ray revealed a fracture of the right parietal bone. On regaining consciousness he was totally aphasic with a partial flaccid paralysis of the right arm and leg.

Examination—The paralysis and the aphasia are now much less, but his speech is rambling.

A. *Hearing*—A moderate perceptive deafness on the right; a 10 db to 20 db greater perceptive deafness on the left.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Fair; Dur. 18 secs.	Hor. to right. *Amp.*—Fair; Dur. 16 secs.
Vert.—Fair; slight pallor and sweat.	Good; pallor and sweat.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—None.	Rot. to right. *Amp.*—Finest possible; after 32 secs.; Dur. 58 secs.
Vert.—None.	None.

HEAD BACK.

Nystagmus—Hor. to right. *Amp.*—Good; irregular.	Obl. to right. *Amp.*—Large, irregular.
Vert.—Fair.	Fair.
Atkinson Ratio—.....	1:1.8.

Summary—Responses to rotation subnormal especially from right tracts; responses to caloric tests absent from right vertical tracts, markedly subnormal from left vertical tracts.

Neurotologic Diagnosis—Marked direct injury to upper brain stem especially on right side with contrecoup injury to left cerebral and cerebellar cortex.

Neurosurgical Diagnosis—Same.

CASE NO. 44: N. C. J., age 34. Admission date March 20, 1955. Examined March 25, 1955. Reg. No. 56186.

History: This patient has been subject to grand mal attacks for several years. He was sent to our department as a matter of interest in our findings.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—Except a large vertical nystagmus on upward gaze all are within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 22 secs.	Hor. to right. *Amp.*—Good; Dur. 19 secs.
Vert.—Fair; no autonomic phenomena.	Fair; pallor, sweat and slight nausea.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Fair; after 35 secs.; Dur. 82 secs.	Rot. to right. *Amp.*—Good; after 20 secs.; Dur. 91 secs.
Vert.—Marked pallor and sweat.	Good.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Small.	Hor. to right. *Amp.*—Good.
Vert.—Fair.	Good.
Atkinson Ratio—1:2.3.	1:4.5.

Summary—Responses to rotation are just within normal limits; caloric tests show marked depression in response from right tracts, the left being normal.

Neurotologic Diagnosis—High localized lesion on the right.

Neurologic Diagnosis—Grand mal with focus in the right temporal lobe.

CASE No. 5: E. H., age 25. Admitted Feb. 10, 1952. Examined March 7, 1952. Reg. No. 34020.

History: On January 18, 1951, this patient underwent brain surgery. A medullo-blastoma was removed subtotally from the right cerebellar hemisphere. This was followed by X-ray treatment with clinical improvement. This case was sent to us for calibration of our tests.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—A vertical nystagmus on upward gaze. Otherwise within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 21 secs.	Hor. to right. *Amp.*—Good, with rapid decline; Dur. 15 secs.
Vert.—None.	None.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Fair; after 26 secs.; Dur. 107 secs.	Rot. to right. *Amp.*—Good; after 26 secs.; Dur. 102 secs.
Vert.—Marked; pallor, sweat and emesis.	Fair.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—Good.	Fair.
Atkinson Ratio—1:4.1.	1:3.9.

Summary—Response to rotation, left canals normal; right canals depressed; response to caloric test, all canals respond normally.

Neurotologic Diagnosis—Low lesion on right.

CASE No. 40: W. O'N., age 43. Admitted Feb. 10, 1955. Examined Feb. 18, 1955. Reg. No. 55500.

History: Staggering and stumbling to right and to left without vertigo for five years. He is unable to walk or to stand in the dark. No tinnitus or hearing loss.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—First degree nystagmus in both directions, greater on right gaze. On walking test he deviated and staggered 5 feet to right. Rombergism present, falling to right with all head positions.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 36 secs.	Hor. to right. *Amp.*—Good; Dur. 36 secs.
Vert.—Subnormal; no autonomic phenomena.	Subnormal; no autonomic phenomena.

HEAD 120° FORWARD.

Nystagmus—Rot. to left. *Amp.*—Good; Dur. 20 secs.	Rot. to right. *Amp.*—Good; Dur. 24 secs.
Vert.—Subnormal.	Subnormal.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 24 secs.; Dur. 100 secs.	Rot. to right. *Amp.*—Good; after 25 secs.; Dur. 103 secs.
Vert.—Subnormal.	Slbnormal.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—Subnormal.	Subnormal.
Atkinson Ratio—1:4.	1:4.

Summary—Response to rotation. Nystagmus from all canals markedly exaggerated with subnormal vertigo. Response to caloric tests; nystagmus from all canals normal with subnormal vertigo.

Neurotologic Diagnosis—A diffuse or disseminated cerebellar lesion.

Neurologic Diagnosis—1. Multiple sclerosis. 2. Friedrich's Ataxia (degeneration of the pyramidal and cerebellar tracts.)

Neurosurgical Diagnosis—Degeneration of the cerebellar system.

CASE No. 30: G. B. B., age 27. Admitted Oct. 26, 1953. Examined Nov. 20, 1953. Reg. No. 46343.

History: This patient complained only of numbness and progressive "loss of use" of his right side for several months.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—A rotary nystagmus to the right in all five directions of gaze. On walking 20 feet, line deviated to the left and the left arm sagged. Rombergism to the left in all head positions. No compensatory shift of pelvic girdle in any direction.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—large; Dur. 43 secs.	Hor. to right. Amp.—Large; Dur. 35 secs.
Vert.—None.	Markedly subnormal.

HEAD 120° FORWARD.

Nystagmus—Rot. to left. *Amp.*—Good; Dur. 12 secs.	Rot. to right. *Amp.*—Good; Dur. 45 secs.
Vert.—Little or none.	Little or none.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 25 secs.; Dur. 105 secs.	Rot. to right. *Amp.*—Good; After 22 secs.; Dur. 95 secs.
Vert.—Little or none.	Little or none.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—Little or none.	Little or none.
Atkinson Ratio—1:4.2.	1:4.5.

Summary—Response to rotation; nystagmus from all canals except left verticals is markedly exaggerated with subnormal vertigo. Response to caloric tests; nystagmus from all canals within normal limits with subnormal vertigo.

Neurotologic Diagnosis—A disseminated low central lesion.

Neurosurgical Diagnosis—Chronic brain syndrome (syringobulba?).

CASE No. 37: L. V. W., age 22. Admitted Dec. 13, 1954. Examined Dec. 17, 1954. Reg. No. 54235.

History: This case, of utmost clinical significance in the series reported complained of a sensation of heat and nausea, especially on standing; staggering, vomiting, severe occipital headache and weakness, and difficulty in writing with the right hand and arm, of two weeks' duration, and markedly progressive. There was slurring of speech evident only on the attempt to say "Methodist Episcopal" or "Round the rugged rock, etc."

Examination—Eye grounds normal; spinal fluid pressure normal.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—There was a first degree nystagmus in both directions on lateral gaze, greater on looking to the right; a marked vertical nystagmus on upward gaze; during the Romberg test he fell backward in all three positions of the head, and the pelvis was rigid in all directions.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Large; Dur. 38 secs.; swinging.	Hor. to right. *Amp.*—Large; Dur. 36 secs.
Vert.—Good. Pallor, emesis, profuse sweat.	Good; pallor, emesis, profuse sweat.

HEAD 120° FORWARD.

Nystagmus—Rot. to left. *Amp.*—Small; Dur. 20 secs.	Rot. to right. *Amp.*—Small; Dur. 19 secs.
Vert.—None.	Poor.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Large; after 17 secs. Dur. 107 secs.	Rot. to right. *Amp.*—Large; After 15 secs. Dur. 84 secs.
Vert.—Marked; no autonomic phenomena.	Marked; no autonomic phenomena.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Large.	Hor. to right. *Amp.*—Large.
Vert.—Poor.	Poor.
Atkinson Ratio—1:6.3.	1:5.6.

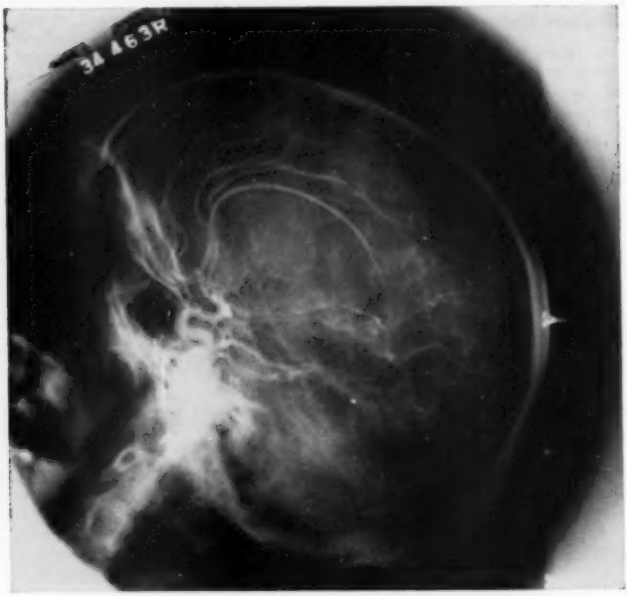

Fig. 6. Case No. 37. Arteriogram showing vascular displacement typical of hydrocephalus.

Summary—All nystagmus responses are markedly exaggerated; the vertigo in the horizontal tracts is abnormal to rotation; the nystagmus response from all tracts is hyperactive, whereas the vertigo is subnormal to the caloric test.

Neurotologic Diagnosis—Advanced lesion in the right cerebellum with extension upward, either infiltration or by encroachment on the foramina

of Magendie and Luschka, with beginning increase of intraventricular pressure.

Neurosurgical Diagnosis—A central nervous system lesion probably cerebellar, but exact site as yet undetermined.

Subsequent History—On Dec. 22 the optic discs became blurred, and the headache became much worse. Arteriograms showed vascular displacement typical of hydrocephalus.

On Dec. 23 his condition became much worse with convulsions and unconsciousness. Immediate surgery revealed "an astrocytoma including the entire right cerebellar system, which appeared to extend to the brain stem." "Marked hydrocephalus." The patient expired on the table.

CASE No. 46: C. W. D., age 39. Admitted April 25, 1955. Examined May 6, 1955. Reg. No. 57118.

History: This patient was sent from neurosurgery for calibration of our tests without previous history. The only history we had was of staggering, double vision, headache and vomiting for several weeks, progressive.

EXAMINATION.

Paralysis of the right VIth cranial nerve, and marked slurring of speech.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—A second degree rotary nystagmus on forward gaze, a marked vertical nystagmus on upward gaze, and on oblique nystagmus up and to the left, with the head in the back horizontal position. There was marked staggering and deviation to the left on walking test. Rombergism present to the right and backward in all three head positions. The pelvic girdle reaction was positive, being rigid as a board in attempts to tilt in all four directions.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Large; Dur. 31 secs., swinging.	Hor. to right. *Amp.*—Large; Dur. 33 secs.; swinging.
Vert.—Good; no autonomic phenomena.	Good; no autonomic phenomena.

HEAD 120° FORWARD.

Nystagmus—Rot. to left. *Amp.*—Large; Dur. 19 secs.	Rot. to right. *Amp.*—Tiny; Dur.
Vert.—Poor.	Poor.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left ear.
Nystagmus—Rot. to left. *Amp.*—Large; after 15 secs., swinging.	Rot. to right. *Amp.*—Large; after 25 secs.; swinging.
Vert.—Poor; no autonomic phenomena.	Poor; no autonomic phenomena.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Large; swinging.	Hor. to right. *Amp.*—Large; swinging.
Vert.—Poor.	Poor.
Atkinson Ratio—1:8.	1:5.8.

Summary—1. The spontaneous and positional nystagmus indicates a central lesion; 2. The Romberg and pelvic girdle reactions tell of a cerebellar lesion; 3. The right sixth nerve paralysis denotes a high, right lesion; 4. The slurring speech is indicative of a high pontine lesion; 5. The marked exaggeration of all the nystagmus responses and the lessened to poor vertigo responses signifies primary low lesion, with upward spread by invasion or increase in pressure.

Neurotologic Diagnosis—Primary low (cerebellar) lesion with upward extension by invasion or increase of generalized pressure.

Neurosurgical Diagnosis—Information was now given that this patient had had a double mammectomy for bilateral adenocarcinoma a few months previously. The diagnosis then was metastasis to the cerebellum, with secondary higher metastases.

CASE No. 38: L. L. R., age 33. Admitted Jan. 17, 1955. Examined Jan. 25, 1955. Reg. No. 54903.

History: This patient complained of persistent dizziness for two months, becoming worse two days previously; staggering, nausea and vomiting in spells lasting two to three days, continuous occipital headache and blurred vision for two weeks.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—A marked vertical nystagmus on upward gaze. Staggering and ataxia on walking test. He fell to the left with all positions of the head in the Romberg test, and his pelvis was rigid to tilt in all directions.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 42 secs.	Hor. to right. *Amp.*—Good; Dur. 42 secs.
Vert.—Poor; no autonomic phenomena.	Poor; no autonomic phenomena.

HEAD 120° FORWARD.

Nystagmus—Rot. to left. *Amp.*—Fair.; Dur. 22 secs.	Rot. to right. *Amp.*—Fair; Dur. 17 secs.
Vert.—Poor.	Poor.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Fair; After 22 secs.; Dur. 103 secs.	Rot. to right. *Amp.*—Fair; After 16 secs.; Dur. 114 secs.
Vert.—Fair; no autonomic phenomena.	Fair; no autonomic phenomena.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Fair.	Hor. to right. *Amp.*—Fair; very slow.
Vert.—Fair.	Fair.
Atkinson Ratio—1:4.6.	1:6.7.

Summary—Responses to rotation; marked exaggeration of nystagmus from horizontal tracts and depression of nystagmus from right vertical

tract; vertigo subnormal. Responses to caloric tests; exaggeration of nystagmus from left verticals, the other normal; vertigo subnormal.

Neurotologic Diagnosis—Posterior fossa (cerebellar) lesion expanding upward or increasing intraventricular pressure.

Arteriogram—Anterior bowing and elevation of the right anterior cerebral artery to the right consistent with hydrocephalus.

Neurosurgical Diagnosis—At operation on Jan. 27, 1955, a glioma involving almost the entire left portion of the cerebellum, and probably involving the brain stem.

CASE No. 3: J. R. M., age 30. Admitted Oct. 25, 1951. Examined Jan. 11, 1952. Reg. No. 40391.

History: This patient was admitted to the ophthalmologic department with ophthalmoplegia involving all three nerves bilaterally. It was diagnosed Guillain-Barre's syndrome, involving the eye muscle nuclei; cause unknown. There was no benefit from the use of antibiotics and ACTH. He was readmitted on Jan. 9, 1952, for a check-up. There was improvement with only residual bilateral involvement of both lateral recti muscles. It is believed that the disorder was of vascular origin.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 42 secs.	Hor. to right. *Amp.*—Good; Dur. 26 secs.
Vert.—Good; little or no autonomic phenomena.	Subnormal; slight nausea; no sweat.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Poor; After 32 secs.; Dur. 103 secs.	Rot. to right. *Amp.*—Poor; after 27 secs.; Dur. 94 secs.
Vert.—Good; nausea, sweat and pallor.	Good; nausea, sweat and pallor.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Fair.	Hor. to right. *Amp.*—Good.
Vert.—Good.	Good.
Atkinson Ratio—1:3.2.	1:3.4.

Summary—Responses to rotation; right tracts normal; left tracts exaggeration. Responses to caloric tests; bilateral depression.

Neurotologic Diagnosis—Primary high central lesion with irritation downward on left.

CASE No. 4: R. J. M., age 32. Admitted Dec. 26, 1951. Examined Jan. 1, 1952. Reg. No. 37713.

History—This patient entered Hines Hospital in 1947 complaining of "black-out" spells, severe headache, nausea and vomiting. At that time

an ependymoma was subtotally removed from the fourth ventricle. He entered this hospital complaining of recurrent severe headache.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—A marked second degree horizontal nystagmus to the left, changing to vertical on upward gaze. He past pointed with both hands three inches to the left, staggered markedly on walking and fell backward with all positions of the head in the Romberg test; pelvis rigid.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Rot. to left. *Amp.*—Huge; Dur. 61 secs.	Rot. to right. *Amp.*—Large; Dur. 35 secs.
Vert.—Little or none; no autonomic phenomena.	Little or none; no autonomic phenomena.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Large; after 16 secs.; Dur. 103 secs.	Rot. to right. *Amp.*—Large; After 12 secs.; Dur. 119 secs.
Vert.—None.	None.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Huge.	Hor. to right. *Amp.*—Huge.
Vert.—None.	None.
Atkinson Ratio—1:6.4.	1:10.

Summary—The responses to both rotation and caloric tests are markedly exaggerated. Since we know the site of the original lesion we may trace its subsequent invasion, both upward and downward, with probable increase of pressure. On March 31, 1953, he was seen in the terminal stage, stuporous, incontinent and with left hemiplegia.

CASE No. 14: W. L. M., age 28. Admitted Oct. 1, 1952. Examined Oct. 3, 1952. Reg. No. 38288.

History: In July, 1951, this patient noticed diplopia on looking to the left. This soon became permanent in all positions of gaze. In January, 1952, at biopsy a chondrosarcoma of the left sphenoid ridge was found. At the same time diabetes insipidus was diagnosed. In September, 1952, he developed blindness of the left eye with severe generalized left hemicrania and weakness of the right arm and leg.

EXAMINATION.

Paralysis left external rectus muscle.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 34 secs.	Hor. to right. *Amp.*—Good; Dur. 40 secs.
Vert.—Subnormal; no autonomic phenomena.	Subnormal; no autonomic phenomena.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. Amp.—Huge; after 15 secs.; Dur. 110 secs.	Rot. to right. *Amp.*—Huge; After 16 secs.; Dur. 114 secs.
Vert.—Fair.	Fair.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Huge.	Hor. to right. *Amp.*—Huge.
Vert.—Fair.	Fair.
Atkinson Ratio—1:7.3.	1:7.1.

Summary—The original site of this lesion is known to be high on the left. Its progress upward and down to the posterior fossa can be traced in these tests. Responses to rotation are exaggerated, especially from the right tracts; those from the caloric tests are most markedly exaggerated.

CASE No. 19: T. A. P., age 28. Admitted Nov. 5, 1952. Examined Nov. 21, 1952. Reg. No. 39077.

History: This is a case of grand mal of nine years' duration, with increasingly frequent typical generalized convulsions with aura.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Huge; Dur. 45 secs.	Hor. to right. *Amp.*—Huge; Dur. 37 secs.
Vert.—Fair; no autonomic phenomena.	Fair; no autonomic phenomena.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 20 secs.; Dur. 114 secs.	Rot. to right. Amp.—Good; After 20 secs.; Dur. 113 secs.
Vert.—Poor; no autonomic phenomena.	Poor; no autonomic phenomena.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—Poor.	Poor.
Atkinson Ratio—1:5.7.	1:5.6.

Summary: Responses to both rotation and caloric tests show exaggeration. Since the original site of this disturbance is known to be high, the spread of the irritation downward can be traced.

CASE No. 29: G. A., age 59. Admitted Nov. 5, 1953. Examined Nov. 13, 1953. Reg. No. 46310.

History: The complaint in this case was frontal headache and failing vision for one year. On examination there was bilateral tubular vision only.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Rot. to left. *Amp.*—Huge; Dur. 32 secs.	Rot. to right. *Amp.*—Large; Dur. 32 secs.
Vert.—Subnormal.	Subnormal.

HEAD 120° FORWARD.

Nystagmus—Rot. to left. *Amp.*—Good; Dur. 15 secs.	Rot. to right. *Amp.*—Good; Dur. 19 secs.
Vert.—Fair; nausea.	Good; nausea and emesis.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Large; After 20 secs.; Dur. 160 secs.	Rot. to right. Amp.—Large; after 16 secs.; Dur. 158 secs.
Vert.—Marked nausea, sweat and emesis.	Marked nausea, pallor and emesis.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Huge.	Hor. to right. *Amp.*—Large.
Vert.—Good.	Good.
Atkinson Ratio—1:8.	1:8.3.

Summary—Response to rotation; moderate exaggeration; response to caloric tests;; marked exaggeration.

Neurotologic Diagnosis—A high midline lesion expanding with increased pressure to posterior fossa.

Neurosurgical Diagnosis—On Nov. 16, 1953, a large sellar pituitary tumor was removed by right transfrontal craniotomy.

CASE No. 32: D. R., age 31. Admitted March 30, 1954. Examined April 23, 1954. Reg. No. 49020.

History: This patient presents diplopia; slight dizziness; ringing in the right ear, and low grade fever (99=100 F.) with sudden onset two weeks previously.

EXAMINATION.

Paralysis of the left rectus muscle. All other findings, both neurological and neurosurgical, were normal.

A. *Hearing*—The bone conduction and air conduction of both ears were approximately identical (all four curves) and were as follows: Dips to

10 db at 1,000 cps., to 30 db at 2,000 cps., to 40 db at 4,000 cps., to 25 db at 4,000 cps., and to 55 db at 8,000 cps.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Huge; Dur. 31 secs.	Hor. to right. *Amp.*—Huge; Dur. 42 secs.
Vert.—Subnormal; no autonomic phenomena.	Fair; no autonomic phenomena.

HEAD 120° FORWARD.

Nystagmus—Rot. to left. *Amp.*—Fair; Dur. 14 secs.	Rot. to right. *Amp.*—Large; Dur. 22 secs.
Vert.—Marked; slight sweat.	Marked; slight sweat.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 28 secs.; Dur. 98 secs.	Rot. to right. *Amp.*—Large; After 32 secs.; Dur. 128 secs.
Vert.—Good; marked pallor and sweat.	Good; pallor and sweat.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Large.
Vert.—Good.	Good.
Atkinson Ratio—1:3.5.	1:4.

Summary—Marked exaggeration of responses of right tracts to rotation with less exaggeration from the left tracts; slight depression of the left tracts and normal response of the right tracts to caloric stimulation.

Neurotologic Diagnosis—An expanding lesion, high, involving the left abducens nucleus and the right vestibular nuclei, with extension by pressure or infiltration to posterior fossa, or a disseminated lesion, both high and low, with first involvement high.

Medical Diagnosis—Multiple sclerosis.

CASE No. 43: J. J., age 34. Admitted March 16, 1955. Examined March 18, 1955. Reg. No. 56193.

History: A few weeks ago this patient had a posterior fossa craniotomy at another hospital. Prior to and since that time he has had trouble with walking, failing vision, and lately some reduction in hearing acuity.

EXAMINATION.

A. *Hearing*—A slight across-the-board reduction of bilateral perceptive type.

B. *Spontaneous Phenomena*—A large vertical nystagmus on upward gaze, past-pointing six inches to the left with the left hand, deviation to the right with ataxia and stumbling forward on walking, Rombergism to the left with all positions of the head, and rigidity of the pelvis on tilting.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Huge; Dur. 74 secs.	Hor. to right. *Amp.*—Large; Dur. 60 secs.
Vert.—Marked; no autonomic phenomena.	Marked—No autonomic phenomena.

HEAD 120° FORWARD.

Nystagmus—None.	Rot. to right. *Amp.*—Large; Dur. 20 secs.
Vert.—None.	Fair.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Large; after 20 secs.; Dur. 150 secs.	Rot. to right. *Amp.*—Large; After 15 secs.; Dur. 119 secs.
Vert.—None.	None.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Large.	Hor. to right. *Amp.*—Large.
Vert.—None.	None.
Atkinson Ratio—1:7.5.	1:8.

Summary—The spontaneous phenomena are those of a cerebellar lesion. All induced phenomena, both rotational and caloric, are exaggerated.

Neurotologic Diagnosis—The original posterior fossa lesion now shows extension upward, either from extension of the lesion or increase of the ventricular pressure.

Arteriogram—This shows a hemangioma of the posterior fossa with multiple dilated aneurisms of the entire circle of Willis.

CASE No. 47: E. D., age 59. Admitted May 4, 1955. Examined May 6, 1955. Reg. No. 57151.

History: This patient complained of difficulty with his vision, blurring and diplopia, and inability to look upward, with unsteadiness in walking. There was no nausea, headache or dizziness. The duration was about three weeks.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—A rotary nystagmus up and to the left in all directions of gaze except that upward gaze was impossible; he deviated markedly to the right on walking and on the Romberg test he fell backward when the head was turned to the left.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Large; Dur. 34 secs.	Hor. to right. *Amp.*—Large; Dur. 35 secs.
Vert.—Good; slight nausea.	Good; slight nausea.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Large; After 26 secs.; Dur. 125 secs.	Rot. to right. Amp.—Large; After 18 secs.; Dur. 133 secs.
Vert.—Fair.	Good; nausea and sweat.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Large.	Hor. to right. *Amp.*—Large.
Vert.—Fair.	Good.
Atkinson Ratio—1:4.8.	1:7.4.

Summary—There is exaggeration of all responses to both rotation and caloric tests, showing irritation extending upward and downward from the tentorium, probably from a point just above the tentorium, as indicated by his inability to gaze upward with both eyes.

Neurotologic Diagnosis—A lesion at or just above the tentorium, causing irritation both upward and downward.

Roentgenographic Studies—No deviation from the normal.

Neurosurgical Diagnosis—Damage to mesencephalon, locally destructive and not space occupying.

CASE No. 27: S. H., age 37. Admitted Nov. 3, 1953. Examined Nov. 6, 1953. Reg. No. 45889.

History—No history was furnished us at the time of our examination except that the patient had a left side high tumor operated upon in 1945.

EXAMINATION.

A left moderate exophthalmos was apparent.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—A vertical nystagmus on upward gaze. Otherwise all within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 42 secs.	Hor. to right. *Amp.*—Good; Dur. 59 secs.
Vert.—Moderate; no autonomic phenomena.	Moderate; no autonomic phenomena.

HEAD 120° FORWARD.

Nystagmus—Rot. to left. *Amp.*—Good; Dur. 20 secs.	Rot. to right. *Amp.*—Good; Dur. 18 secs.
Vert.—Fair.	Subnormal.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Large; After 18 secs.; Dur. 129 secs.	Rot. to right. *Amp.*—Large; After 17 secs.; Dur. 110 secs.
Vert.—Subnormal.	Subnormal.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Large.	Hor. to right. *Amp.*—Large.
Vert.—Subnormal.	Subnormal.
Atkinson Ratio—1:7.1.	1:6.4.

Summary—All nystagmus responses to both rotation and the caloric tests are exaggerated. The vertigo responses are fairly normal. The latter finding indicates that the cerebellum is secondarily involved.

Neurotologic Diagnosis—A primary high lesion with secondary irritation of the posterior fossa structures.

Neurosurgical Diagnosis—Meningioma recurrent of left middle and frontal fossae, and left intraorbita. Generalized chronic brain syndrome associated with the neoplasm.

CASE No. 11: M. W. H., age 34. Admitted April 29, 1952. Examined May 23, 1952. Reg. No. 35249.

History—This patient had acute meningo-encephalitis in 1946. Since then he has complained of severe head and neck pains, with a series of other vague complaints such as marked irritability, loss of libido, etc.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—There was consistent past-pointing to the left of two to three inches, Rombergism was present with sway to the left in all head positions; he was unable to complete the walking test because of ataxia and toppling to the left; the pelvis was rigid to the tilt test in all directions.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Large; Dur. 61 secs.	Hor. to right. *Amp.*—Large; Dur. 41 secs.
Vert.—Subnormal; no autonomic phenomena.	Subnormal; autonomic phenomena subnormal.

HEAD 120° FORWARD.

Nystagmus—Rot. to left. *Amp.*—Good; Dur. 30 secs.	Rot. to right. *Amp.*—Good; Dur. 25 secs.
Vert.—Fair.	Good; Nausea and emesis.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 20 secs.; Dur. 121 secs.	Rot. to right. *Amp.*—Good; After 25 secs.; Dur. 120 secs.
Vert.—Fair.	Fair.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—Fair.	Fair.
Atkinson Ratio—1:6.	1:4.8.

Summary—Both rotation and the caloric tests showed marked hyperirritability of both vestibular mechanisms, but the picture is irregular;

e.g., the right side shows greater response to rotation than to the caloric tests, whereas the left side shows greater response to the caloric tests than to rotation. The sense of vertigo is markedly subnormal, except that after rotation the right vertical canal mechanism showed hypersensitivity. (Here the vestibular or canal mechanism includes both the end-organ and the central tracts and connections.)

Neurotologic Diagnosis—A widely disseminated lesion of an irritative nature in accord with residual effects of meningo-encephalitis of the entire brain.

Neurosurgical Diagnosis—Chronic brain syndrome (post encephalitic).

Case No. 12: W. P., age 63. Admitted May 31, 1952. Examined July 11, 1952. Reg. No. 35921.

History: This patient complained of fainting spells, dizziness and tinnitus over the period of the past six or seven years. His other symptoms and findings were typical of hypertension, the radial blood pressure being 260/104.

EXAMINATION.

A. *Hearing*—The audiogram indicated a typical progressive senile perceptive deafness.

B. *Spontaneous Phenomena*—There was marked ataxia and staggering, Rombergism was present with falling to the left with all head positions, and the pelvis was rigid to the tilt test in all directions.

Head 30° Forward.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Huge; Dur. 21 secs.	Hor. to right. *Amp.*—Large; Dur. 25 secs.
Vert.—Fair; little or no autonomic phenomena.	Marked with oscillations of the head and body, especially to right. Emesis.

Head 30° Forward.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 40 secs.; Dur. 85 secs.	Rot. to right. *Amp.*—Large; After 25 secs.; Dur. 95 secs.
Vert.—Fair; slight nausea.	Marked; nausea and emesis.

Head Back.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Huge.
Vert.—Fair to good.	Marked.
Atkinson Ratio—1:2.1.	1:3.8.

Summary—The responses to rotation are within normal limits except for the bizarre oscillations of the head and body indicating cerebellar irritation. The caloric responses are subnormal, indicating damage to the higher (brain stem and cerebral) tracts.

Neurotologic Diagnosis—A generalized arteriosclerotic condition of the central tracts.

On May 15, 1954, this patient expired in this hospital.

Anatomic Diagnosis—Myocardial infarction; arteriosclerosis of the central nervous system.

CASE No. 17: B. R. M., age 32. Admitted Oct. 31, 1952. Examined Nov. 7, 1952. Reg. No. 38977.

History: The complaints were paraesthesia of the right little finger and of taste on the left side of the tongue; unsteadiness of gait with staggering, inability to tell just where his feet are in space with inability to make the left foot obey his will, and transient diplopia; all for about six months; no headache or convulsions.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—Staggered and deviated six feet to the right on walking test, and Rombergism present as a sway with falling to the right on turning the head to the right.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Huge; Dur. 63 secs.	Hor. to right. *Amp.*—Large; Dur. 55 secs.
Vert.—Subnormal.	Fair; slight pallor and sweat.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Large; After 25 secs.; Dur. 110 secs.	Rot. to right. *Amp.*—Good; After 20 secs.; Dur. 102 secs.
Vert.—Marked; nausea, sweat, pallor, emesis.	Marked; nausea and emesis.

HEAD BACK.

Nystagmus—Hor to left. *Amp.*—Large.	Hor. to right. *Amp.*—Large.
Vert.—Marked.	Marked.
Atkinson Ratio—1:4.4.	1:5.1.

Summary—All responses to rotation and the caloric tests are exaggerated, the least being from the right vestibular system.

Neurotologic Diagnosis—A widely disseminated central disturbance.

Neurosurgical and Medical Diagnosis—Multiple sclerosis.

CASE No. 22: W. P., age 33. Admitted March 12, 1953. Examined March 13, 1953. Reg. No. 42760.

History: There was a muscle weakness of the entire right side and a generalized numbness of two months' duration; it is now improving. Otherwise his history and general physical examination are negative.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—There was crossed past-pointing of both hands and persistent falling to the left with all positions of the head in the Romberg test.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 32 secs.	Hor. to right. *Amp.*—Good; Dur. 33 secs.
Vert.—Good; pallor, sweat and nausea.	Good; marked pallor, sweat and nausea.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 35 secs.; Dur. 98 secs.	Rot. to right. *Amp.*—Good; After 27 secs.; Dur. 107 secs.
Vert.—Good; pallor, sweat and emesis.	Good; pallor, sweat and emesis.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—Good.	Good.
Atkinson Ratio—1:2.8.	1:4.

Summary—Responses of all canals to rotation are exaggerated; the right system is markedly depressed, and the left system is normal to the caloric tests. These findings, together with the spontaneous phenomena, indicate generalized involvement with reversible damage high on the right.

Neurotologic Diagnosis—A reversible (probably vascular) generalized disturbance most especially high on the right.

Neurologic and Neurosurgical Diagnosis—A diffuse central lesion, probably vascular.

CASE No. 24: B. J. B., age 26. Admitted April 17, 1953. Examined May 1, 1953. Reg. No. 42239.

History: For three years this patient had severe occipital headache with a swimming sensation, most marked on coughing or sneezing.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 40 secs.	Hor. to right. *Amp.*—Good; Dur. 46 secs.
Vert.—Fair; profuse sweat.	Fair; profuse sweat.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 22 secs.; Dur. 114 secs.	Rot. to right. *Amp.*—Large; After 21 secs.; Dur. 119 secs.
Vert.—Little or none.	Little or none.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Large.	Hor. to right. *Amp.*—Large.
Vert.—Little or none.	Little or none.
Atkinson Ratio—1:5.2.	1:5.7.

Summary—All responses to both rotation and the caloric tests are markedly exaggerated.

Neurotologic Diagnosis—A diffuse disturbance involving both the high and the low central nervous system.

Neurosurgical and Roentgenographic Diagnosis—Platybasia.

CASE No. 39: M. G., age 45. Admitted Dec. 28, 1954. Examined Jan. 28, 1955. Reg. No. 54475.

History: This patient complained of generalized pains in body and limbs (on previous admission to this hospital he had complained of various vague and undiagnosed body pains), inability to step up on curb, and staggering. A few months previously, on turning his head to the right there was a sudden pain as of an electric shock running up the right side of his head with flashing of red lights in front of his eyes, staggering and stumbling, but without true vertigo.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—Deviation and staggering to the right, Rombergism with falling to the right with all head positions and rigidity of the pelvis to tilting in all directions.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Large; Dur. 55 secs.	Hor. to right. *Amp.*—Good; Dur. 34 secs.
Vert.—Poor; no autonomic phenomena.	Poor; no autonomic phenomena.

HEAD 120° FORWARD.

Nystagmus—Rot. to left. *Amp.*—Large; Dur. 26 secs.	Rot. to right. *Amp.*—Good; Dur. 20 secs.
Vert.—Poor and perverted.	None.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 20 secs.; Dur. 129 secs.	Rot. to right. *Amp.*—Good; After 11 secs.; Dur. 123 secs.
Vert.—None.	None.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Large; swinging.	Hor. to right. *Amp.*—Huge; swinging.
Vert.—None.	None.
Atkinson Ratio—1:6.4.	1:11.

Summary—All responses to both rotation and the caloric tests are markedly exaggerated, especially from the left system.

Neurotologic Diagnosis—A widespread disseminated disease of the central nervous system primary or most advancement in the cerebellum.

Neurologic and Neurosurgical Diagnosis—Parenchymatous cortical cerebellar (and cerebral) atrophy.

CASE No. 42: L. S. H., age 43. Admitted March 1, 1955. Examined March 11, 1955. Reg. No. 55878.

History: This case was sent to the otological department without a history for calibration of our tests. Our evaluation was wanted also.

EXAMINATION.

The patient was morose and introspective during the examination.

A. *Hearing*—A mild across-the-board decrease of hearing acuity bilateral, especially on the left of perceptive type without recruitment or diplacusis.

B. *Spontaneous Phenomena*—A deviation of seven feet on the walking test, Rombergism, with falling to the left with all head positions, and rigidity of the pelvis to tilting in all directions.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Large; Dur. 19 secs.	Hor. to right. *Amp.*—Large; Dur. 15 secs.
Vert.—Fair; no autonomic phenomena.	Fair; no autonomic phenomena.

HEAD 120° FORWARD.

Nystagmus—Rot. to left. *Amp.*—Good; Dur. 17 secs.	Rot. to right. *Amp.*—Good; Dur. 15 secs.
Vert.—Fair.	Fair.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Tiny; After 43 secs.; Dur. 73 secs.	Rot. to right. *Amp.*—Small; After 37 secs.; Dur. 90 secs.
Vert.—Little or none.	Little or none.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Very small.	Hor. to right. *Amp.*—Good.
Vert.—Little or none.	Little or none.
Atkinson Ratio—1:1.7.	1.2.4.

Summary—All responses from both rotation and the caloric tests are impaired from both vestibular systems, especially the right.

Neurotologic Diagnosis—A widely disseminated central (supra- and infra-tentorial) nervous system organic disease with psychic overlay.

Neurosurgical Diagnosis—Multiple sclerosis (?).

Medical Diagnosis—Psychosis.

CASE No. 8: J. D. G., age 25. Admitted March 10, 1952. Examined April 4, 1952. Reg. No. 34203.

History: Recurrent pain over the distribution of the maxillary division of the right fifth cranial nerve typical of tic douloureaux. He has had surgery of the mandibular branch, and evulsion of the maxillary branch with recurrence of the pain.

EXAMINATION.

A. *Hearing*—A slight across-the-board lowering of the threshold for acuity of the perceptive type.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. Amp.—Fair; Dur. 14 secs.	Hor. to right. *Amp.*—Fair; Dur. 10 secs.
Vert.—Subnormal.	Subnormal.

HEAD 120° FORWARD.

Nystagmus—Rot. to left. *Amp.*—Good; Dur. 14 secs.	Rot. to right. *Amp.*—Fair; Dur. 11 secs.
Vert.—Marked; emesis.	Marked; emesis.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Small; After 35 secs.; Dur. 86 secs.	Rot. to right. *Amp.*—Small; After 37 secs.; Dur. 86 secs.
Vert.—Subnormal; emesis.	Subnormal; emesis.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Large.
Vert.—Fair; emesis.	Fair; emesis.
Atkinson Ratio—1:2.4.	1:2.3.

Summary—All responses to rotation and to the caloric tests are markedly subnormal except those from the left vertical canal system.

Neurotologic Diagnosis—A disturbance (degenerative?) of the entire central nervous system—both high and low.

Neurosurgical Diagnosis—Tic douloureaux. No other pathology found.

CASE No. 13: D. E. W., age 21. Admitted Sept. 14, 1952. Examined Sept. 21, 1952. Reg. No. 35818.

History: This patient complained of uncontrollable sleeping. He was courtmartialed twice while in service for sleeping on post. He now falls asleep while at work. At one time he slept continuously for 43 hours. This was sleep and not coma. True narcolepsy has been ruled out.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 24 secs.	Hor. to right. *Amp.*—Good; Dur. 21 secs.
Vert.—Marked; pallor and profuse sweat.	Marked; pallor and profuse sweat.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Poor; After 56 secs.; Dur. 76 secs.	Rot. to right. *Amp.*—Poor; After 55 secs.; Dur. 65 secs.
Vert.—Marked; pallor, nausea and sweat.	Marked; pallor, nausea and sweat.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Fair.	Hor. to right. *Amp.*—Fair.
Vert.—Good.	Good.
Atkinson Ratio—1:1.4.	1:1.2.

Summary—Responses to rotation normal; responses to caloric tests markedly subnormal.

Neurotologic Diagnosis—A high diffuse lesion.

Neurosurgical and Neuropsychiatric Diagnosis—Conversion hysteria.

CASE No. 15: G. T. M., age 52. Admitted Sept. 30, 1952. Examined Oct. 3, 1952. Reg. No. 38344.

History: For the past two and one-half years this patient had recurrent attacks of dizziness, lightheadedness, nausea and diplopia when under stress.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—Except stiffness of his pelvis to the tilting tests all were within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Huge; Dur. 33 secs.	Hor. to right. *Amp.*—Good; Dur. 26 secs.
Vert.—Marked (fell to right during the turning) pallor and nausea.	Good (slumped in chair during the turning); pallor and nausea.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 28 secs.; Dur. 95 secs.	Rot. to right. *Amp.*—Good; After 22 secs.; Dur. 91 secs.
Vert.—Good; emesis.	Good; emesis.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—Good.	Good.
Atkinson Ratio—1:3.4.	1:4.1.

Summary: Responses to rotation are exaggerated especially from the left system; responses to the caloric tests are nearly normal, being only slightly reduced from the right system.

Neurotologic Diagnosis—A midline cerebellar lesion, more to the left hemisphere, and with early secondary effects on the higher nervous system.

Neurosurgical and Neuropsychiatric Diagnosis—Shortly after the neurotologic examination and while still hospitalized this patient developed a typical paranoid schizophrenic syndrome and so diagnosed.

CASE No. 21: W. C., age 47. Admitted Dec. 19, 1952. Examined Dec. 26, 1952. Reg. No. 39907.

History: This patient has a long history of hospitalizations for vagotomy, subtotal gastrectomy, and for closure of an oro-antral fistula with severe pain in his left cheek. To control pain he requires codein grs. ss every three hours. There may be drug addiction. He has glycosuria of unknown origin. His left eye turns in, which he states followed an injection of his left sphenopalatine ganglion with phenol, which was done recently in an effort to control his pain.

EXAMINATION.

There is a marked weakness, almost a paralysis, of the left external rectus muscle.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 22 secs.	Hor. to right. *Amp.*—Good; Dur. 20 secs.
Vert.—Subnormal.	Subnormal.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 45 secs.; Dur. 72 secs.	Rot. to right. *Amp.*—Good; After 38 secs.; Dur. 92 secs.
Vert.—Poor.	Poor.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—None.	None.
Atkinson Ratio—1:1.6.	1:2.4.

Summary—Responses to rotation are normal; those from caloric testing are markedly subnormal.

Neurotologic Diagnosis—A diffuse or disseminated high lesion.

Neurosurgical and Neurologic Diagnosis—No evidence of neuropathy found.

CASE No. 23: W. P. B., age 28. Admitted March 19, 1953. Examined April 10, 1953. Reg. No. 41637.

History—This patient complained of frontal headache and mild convulsive seizures. One year ago he had a carotid ligation to reduce a possible intracranial hemangioma as there is an extensive hemangioma involving the face and bones (frontal) of the skull. He is a chronic alcoholic.

EXAMINATION.

A. *Hearing*—A dip to 35 db in each ear at 4,000 cps. Otherwise within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Good; Dur. 24 secs.	Hor. to right. *Amp.*—Good; Dur. 22 secs.
Vert.—Fair to good.	Fair to good.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Fair; After 37 secs.; Dur. 85 secs.	Rot. to right. *Amp.*—Good; After 41 secs.; Dur. 74 secs.
Vert.—Fair to good; nausea and sweat.	Good; nausea and sweat.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Fair.	Hor. to right. *Amp.*—Good.
Vert.—Fair.	Good.
Atkinson Ratio—1:2.3.	1:1.7.

Summary—Responses to rotation are normal; to caloric tests they are markedly subnormal.

Neurotologic Diagnosis—A probable high organic lesion.

Neurosurgical Diagnosis—There is no evidence of an intracranial growth at this time.

CASE No. 36: A. B. A., age 45. Admitted Nov. 2, 1954. Examined Nov. 5, 1954. Reg. No. 53422.

History: This patient complained of severe headache of three months' duration. Three nights ago he awakened from sleep dizzy and suffocating. He arose from his bed, and on attempting to walk fell backward to the ground. On arising he had severe vertigo, nausea and staggering, all of which are persistent. One week before admittance he had a black-out while at work. One year ago he was struck on the head with an iron beam.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—Marked staggering; the Romberg test showed swaying with forward gaze and backward falling with the head to right or left, and the pelvis was rigid to tilting in all directions.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Huge; Dur. 41 secs.	Hor. to right. *Amp.*—Large; Dur. 36 secs.
Vert.—Good; autonomic phenomena subnormal.	Good; autonomic phenomena subnormal.

HEAD 120° FORWARD.

Nystagmus—Rot. to left. *Amp.*—Large; Dur. 20 secs.	Rot. to right. *Amp.*—Good; Dur. 23 secs.
Vert.—Fair; slight pallor and sweat.	Fair to good; pallor, sweat, nausea.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 25 secs.; Dur. 103 secs.	Rot. to right. *Amp.*—Good; After 21 secs.; Dur. 98 secs.
Vert.—Good; slight pallor, sweat, nausea.	Good; pallor, sweat, nausea, emesis.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—Good.	Good.
Atkinson Ratio—1:4.	1:4.6.

Summary—Responses to rotation are exaggerated; to caloric tests they are normal.

Neurotologic Diagnosis—A lesion of the midline structures of the cerebellum (probably vascular in origin).

Neurosurgical and Neurophychiatric Diagnosis—Hysteria.

CASE No. 41: E. R. M., age 31. Admitted Feb. 21, 1955. Examined Feb. 25, 1955. Reg. No. 55719.

History—In March, 1952, there was fogging and then blindness of the left eye which lasted three weeks, and recovered spontaneously with no known cause. Three months later he noticed tightness of the head with irregular staggering and falling; again with recovery in three weeks. Three weeks ago he suffered attacks of loss of control of the left leg, and he overshot or undershot the mark with his foot, on attempting to step up on a curb, and his left leg would not track with his right leg on making a turn. There was no history of pain or head injury.

EXAMINATION.

A. *Hearing*—Within normal limits.

B. *Spontaneous Phenomena*—All within normal limits.

HEAD 30° FORWARD.

C. *Rotation*—To right.	To left.
Nystagmus—Hor. to left. *Amp.*—Fair; Dur. 38 secs.	Hor. to right. *Amp.*—Good; Dur. 40 secs.
Vert.—Poor; no autonomic phenomena.	Poor; no autonomic phenomena.

HEAD 30° FORWARD.

D. *Caloric*—Right Ear.	Left Ear.
Nystagmus—Rot. to left. *Amp.*—Good; After 24 secs.	Rot. to right. *Amp.*—Good; After 23 secs.
Vert.—Poor; no autonomic phenomena.	Poor; no autonomic phenomena.

HEAD BACK.

Nystagmus—Hor. to left. *Amp.*—Good.	Hor. to right. *Amp.*—Good.
Vert.—Poor.	Poor.
Atkinson Ratio—1:4.3.	1:4.7.

Summary—To rotation; nystagmus exaggerated, vertigo poor; to caloric tests; nystagmus at upper limit of normal, vertigo poor.

Neurotologic Diagnosis—An early lesion (probably vascular) of the cerebellum, midline.

Neurosurgical and Medical Diagnosis—No evidence of neoplasm. No diagnosis made.

CASE No. 20: E. J. G., age 47. Admitted Nov. 12, 1952. Examined Nov. 21, 1952. Reg. No. 39321.

CASE No. 48: B. L. G., age 41. Admitted May 9, 1955. Examined May 6, 1955. First examined June 5, 1953. Reg. No. 57253.

CASE No. 51: G. G., age 56. Admitted June 9, 1955. Examined June 10, 1955. Reg. No. 57879.

These three cases are not being reported in detail because they were lesions of the cerebello-pontine angle, and as such presented their classical syndrome of a complete peripheral lesion on the side of the lesion with blocking of the tracts from the vertical canals on the opposite side. This syndrome, being so diagnostic of the angle lesion, which seems to present the picture of neither a high nor low lesion, removes these cases from the clinical problem considered by the present work. For this reason these cases are removed from the statistical problem also. This limits the number of cases to a total of 49, rather than the actual total of 52.

SUMMARY OF CASE REPORTS.

Certain presumptions may be made reasonably from the evidence presented by these clinical observations. These presumptions are:

1. The tentorium appears to be the geographic dividing line between high and low lying lesions as here presented.

2. When a lesion is high and localized and the equilibratory system is affected, the responses to the cold caloric test are affected, whereas the responses to rotation remain within normal limits.

3. When a lesion is low and localized the reverse syndrome will appear. The responses to rotation are disturbed, and the responses to the cold caloric test remain within normal limits.

4. In either of the above conditions, if the lesion is irritative the responses will indicate hyperactivity; if the lesion is destructive the responses will indicate hypoactivity.

5. As the effects of the lesion spread upward or downward the syndrome changes accordingly. This is quite evident in the case of a low lying lesion producing a block to normal ventricular drainage.

6. A generalized lesion is indicated by the presence of affected responses, both to rotation and to the cold caloric test.

7. At the present writing there must be corroborating neurological or neurosurgical evidence before surgery is entertained, although the neurosurgeon may well be alerted to an impending surgical crisis.

8. The response to either rotation or caloric stimulation that seems to be basically affected is nystagmus. Effects on vertigo, falling or the autonomic phenomena seem to be secondary.

9. A lesion limited to the cerebello-pontine angle seems to be neutral so far as the differentiation considered here is concerned, seeming to have its own syndrome. For this reason the three cases recorded are not considered statistically.

10. On the basis of the observations made here correct localizations were made in 42 of the 49 cases or in 85.7 per cent of the cases observed.

11. As supportive evidence of the clinical observations reported here we have considered a series of cases reported elsewhere.[7] In their text Drs. Jones and Fisher report a series of cases to illustrate the localization of peripheral and central lesions. The pattern considered in this paper was not considered by them. A series of 22 intracranial lesions is reported. These are cases numbered 11 to 32 inclusive. Cases 26 to 32 are angle lesions and so may be excluded. Of the remaining cases one was undiagnosed, leaving 14 cases for consideration. Of these 14 cases, 10 cases (numbered 11, 12, 13, 15, 16, 19,

20, 21, 23 and 25) or 71.4 per cent show the high-low pattern unmistakably.

ANATOMICO-PHYSIOLOGIC CONSIDERATIONS.

In attempting to understand and to explain the evidence presented by the cases reported the following questions are encountered:

1. Do high lying (upper rhombencephalic, mesencephalic, diencephalic or even telencephalic) centers exist for the mediation of equilibratory phenomena, most particularly for nystagmus?

2. Do low lying posterior fossa or cerebellar equilibratory centers having to do with nystagmus exist?

3. Is there any basic difference in the mechanisms of response to rotation and to the cold caloric test?

4. If the answers to these questions are affirmative, can a theoretical mechanism explaining the modus operandi of the high and low syndromes be established?

Let us consider these problems.

Since Breuer[3] in 1875 noted that stimulation of the labyrinth of the pigeon produced eye movements, and Barany[4,5] in 1906 made his classic observations that caloric stimulation of the labyrinth in humans produced true nystagmus, and that this nystagmus lasted longer than the duration of the peripheral stimulus, the existence of connections between the labyrinth and the eye muscle nuclei via the structures of the brain stem has been recognized. The literature since then is so replete with experimental and clinical data proving their existence as to preclude the possibility of presenting a complete bibliography of this phase of the problem in a discussion of this type. It must suffice to state that the anatomic foundation of the reflex arc in question was laid by Ramon y Cajal between 1887 and 1892 and finally reported by him in 1893.[6]

The later development of this phase of the question may be traced by noting the simplicity of the mechanism as conceived by Isaac Jones[7] in 1918 and comparing it to the mechanism

for the eye muscle reflexes as conceived by Lorente de No[8,9,10] in which the direction, the amplitude and the duration of these reflex responses (most especially nystagmus and compensation after unilateral labyrinthine destruction with which we are concerned here) are made a function of the substantia reticulata, a structure that forms one of the pathways between the labyrinth and the eye muscle nuclei. These various pathways have been accepted by anatomists, physiologists and clinicians alike. Lesions involving these tracts to and through the mid-brain, and their connections with the cerebral cortex, the thalamus, the hypo-thalamus and the extra-pyramidal nuclei, are known to have their clinical effects on the process of equilibration.[11] The eye muscle-labyrinthine reflexes (postural and nystagmatic) are most importantly involved in the considerations of this discussion. These structures and pathways are involved, whether the labyrinth be stimulated by rotation or by caloric excitation.[8,9]

The existence of cerebellar centers governing or mediating nystagmus has not been established for as long a time. Such centers were suspected (*e.g.*, Bauer and Leidler[12] noted that the duration of nystagmus was prolonged by a lesion in the vermis), but not until 1921 were they proven experimentally in the rabbit by Hoshino.[13] He noted that tracts connecting the vestibular nerves with the cerebellum had been recognized by "Bechterew, van Gehuchten, Andre Thomas, Edinger, Winkler, Ramon y Cajal, Fuse, etc.," but he felt that any concept of a connection between eye motion and the cerebellum was vague. He stimulated the cerebellum with a Faradic current and noted that with the head in a horizontal position eye deviation occurred to the stimulated side. After bilateral destruction of the labyrinths the same effect was obtained. He also found that stimulation of the vermis increased the intensity of both post rotatory and calorically induced nystagmus if the labyrinths were intact.

In 1951 Cranmer[14] reported a series of experiments working with monkeys. After observing the effects of both stimulation and destruction of the superior and lateral vestibular nuclei, he noted that Faradization of the right side of the

pyramis of the cerebellum produced slow right conjugate deviation (left nystagmus) of the eyes. Stimulation of the left side of the pyramis caused left conjugate deviation (right nystagmus) of the eyes. Destruction of the pyramis on each side reversed these phenomena. The right nucleus fastigii was then stimulated and a left slow conjugate deviation (right nystagmus) of the eyes was noted. Thus he demonstrated an inhibitory action of the pyramis over the nucleus fastigii, which in its turn has an inhibitory action on the vestibular nuclei of the opposite side of the pons.

Mygind in 1953[15] stated that in unilateral labyrinthine destruction, after compensation has taken place, the cerebellum is no longer an inhibitor only. It also becomes an activator as necessity demands.

It should be noted that both Cranmer and Mygind used only position and rotation to effect the alterations in deviation and nystagmus. No caloric tests were mentioned.

Crosby in 1953[16] confirmed the thesis that the nucleus fastigii and the vermis are mediators of nystagmus.

Cawthorn in 1954[17] was of the opinion that when positional nystagmus has a central origin that site is in the midline of the cerebellum (Table I of his paper); however, he notes that a caloric abnormality in a surprisingly high percentage of such cases exists (Table VI), which would seem to devaluate my own observations.

Thus we conclude that there are centers mediating nytagmus in the cerebellum (subtentorial) as well as in the higher (supratentorial) areas.

In 1931 Lorente de No[8] described for the first time the fact that each face of the cristae of the canals, and the entire macule of the utricle are supplied by three types of nerve fibers; that each type has a regional distribution in the cristae and in the macula and that each type ends in a specific type of end-organ (see Figs. 7 and 8). These structures of the cristae and the utricular macula bear a striking resemblance to each other.

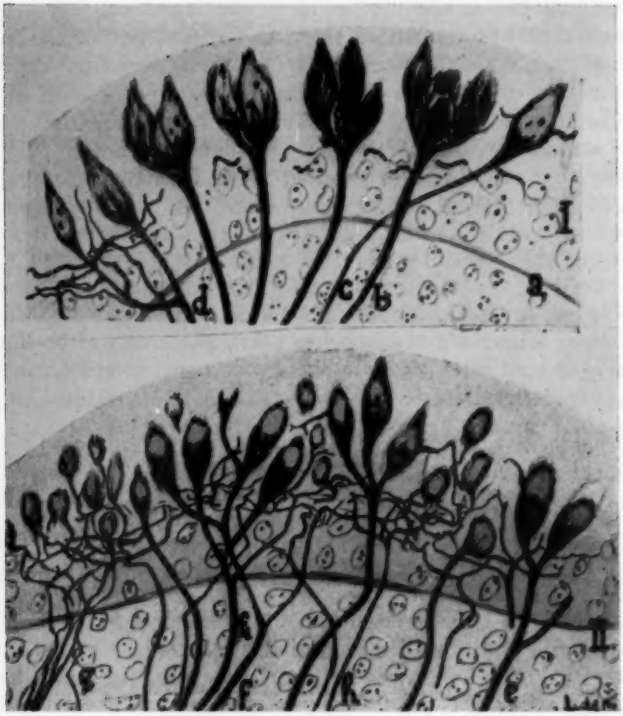

Fig. 7. A cross section of the crista of the anterior vertical canal of a 25-day-old mouse.

I. Central region: b. and c. Thick fibers with single calyces and specific distribution; d. See below.

II. Peripheral region: d., e. and f. Medium thick fibers with branching and multiple calyces showing overlapping distribution; g. and h. Thin fibrils forming an intraepithelial plexus ending in hull-like coating for the hair cells.

The macule of the saccule also contains three somewhat similar but much less differentiated fibers and end-organs (see Fig. 9); furthermore each of the three types of nerve fibers maintains a regional or trunk line grouping within the vestibular nerve, and this specific grouping is maintained by the parent cells within the ganglion of Scarpa.

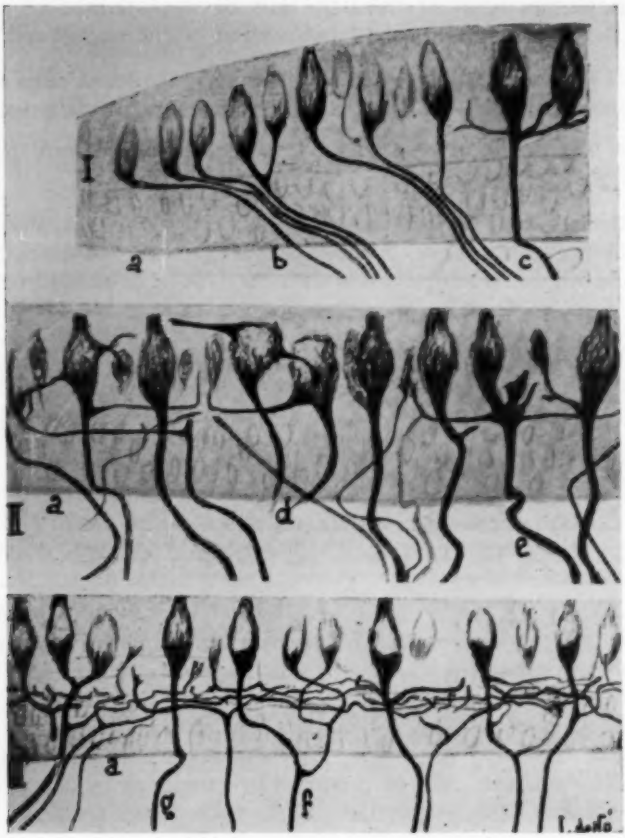

Fig. 8. Cross section of the utricular macula of a 25-day-old mouse: I. Central region; II. Intermediate region; III. Peripheral region.
Note the similarity of the fibers and their endings to those of crista in Fig. 7.

The specificity of these structures surely indicates that each type responds to a specific type of stimulus.

In the same communication Lorente de No points out that the cristae have three possible methods of stimulation. These are:

1. Displacement of the hair cells by displacement of the cupola from momentary current action of the endolymph.

2. Distortion of the cristae by distortive action of currents of the perilymph acting on the enclosed membranous labyrinth.

3. The action of centrifugal force producing outward pull and displacement.

He feels that stimulation of the hair cells of the maculae is produced by alteration in shape of the overlying otolith membrane whose structure varies with the type of end-cells in the

Fig. 9. Cross section of the saccular macula of the rat.
Note on comparison with the structure of the utricular macula in Fig. 8 the markedly lessened degree of differentiation of the types of fibers, the end cells and their distribution.

macula on which it rests. This process is a comparatively slow one when compared with the instant response of the cristae to sudden motion. It may well account for the time lag of postural eye position changes that occurs after experimental ablation of the canal responses. This alteration in shape of the otolith membrane may be produced by gravity or by centrifugal force.

It is evident at once that rotation brings into action all three modalities—hair cell displacement by endolymph deformation of the cristae, distortion of the membranous labyrinth by peri-'ymph currents, and the action of centrifugal force.

It is equally evident that in the caloric tests centrifugal force can play no part. There is then an essential difference in the mechanics of the two processes. There is also the strong probability that the rotational tests as they are performed clinically with high acceleration and long continued duration

may bring into play the maculae, especially the utricular maculae.

Since the answers to the first three of the questions encountered are affirmative, may we assume hypothetically that impulses resulting from centrifugal force have a particular route to the cerebellum, most especially to the pyramis and to the fastigial nucleus; that when a low lying lesion affects these areas of the cerebellum the rotational tests are affected, whereas the caloric tests that do not invoke centrifugal force are unaffected; that per contra when a lesion lying higher (perhaps above the tentorium) does not affect these cerebellar areas the added influence of centrifugal force may allow nystagmus to appear with little or no clinical evidence of alteration of the results of the rotational tests, whereas the caloric tests without this added influence are clinically disturbed.

These are the observations and the deductions of a clinical otologist who is interested in neurotology. It will remain for an experimental scientist in these fields to substantiate or nullify the deductions. The observed clinical phenomena, being factual, should stand while a more nearly correct explanation is sought should the deductions be disproven.

SUMMARY.

1. A syndrome evidenced by high central disturbances and a syndrome evidenced by low lying disturbances of the central nervous system have been described.

2. Their use in differential diagnosis has been indicated.

3. A hypothetical but entirely possible anatomico-physiologic explanation for these syndromes has been presented.

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THE SURGICAL ANATOMY OF THE ANTERO-CAUDAL PORTION OF THE NASAL SEPTUM: A STUDY OF THE AREA OF THE PREMAXILLA.*

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INTRODUCTION.

It is the purpose of this paper to explore the anatomy of the caudal end of the nasal septum with the object of gaining a better understanding of the surgical anatomy. In performing septal surgery, a detailed knowledge of the framework of the premaxillary area is essential. A surgical procedure upon the septum, that requires a thorough understanding of this structure will be presented.

The desired information about the anatomy of the premaxilla and septal operative technique is not available in a single publication. It would seem advantageous to assemble this information of the surgical anatomy of the antero-caudal end of the nasal septum into one dissertation.

The author wishes to express his appreciation for the important groundwork laid for this study while attending seminars presented by Dr. M. H. Cottle.[1]

OBJECTIVES.

A detailed study of cadaver dissections and a report of observations made upon many surgical procedures on the antero-caudal end of the nasal septum will be presented. This is necessary in order to justify an improved operative procedure.

The objectives are:

1. To present a detailed description of the mucoperichon-

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drial and mucoperiosteal reflections of the antero-caudal end of the nasal septum.

2. To assemble pertinent data derived from a study of the rigid structures from this same area on cadavers and operated patients.

3. To emphasize the failure of the standard submucous resection incision to provide clear access to the premaxillary area.

4. To investigate the concept that an intranasal incision and "undermining the base of the nose"[2] provides an improved exposure for correcting caudal basal spurs and deviations of the nasal septum.

NOMENCLATURE.

1. Antero-Caudal End of the Nasal Septum: This is divided into three parts; bony, cartilaginous, and membranous septum. The bony portion of the septum is composed of a part of the premaxilla and its process (anterior nasal spine and premaxillary wing), the crest of the superior maxilla articulating superiorly with the vomer and anteriorly with the premaxillary wing, and the vomer having attachments with the premaxillary wing. This bony portion of the antero-caudal end of the nasal septum will be designated the premaxillary area in this dissertation (see Fig. 1).

The anterior nasal spine is a sharp median spur of bone that projects from the lower margin of the pyriform aperture, and its base is formed by the two maxillae meeting in the midline. Projecting from its base in the midline is a sharp prominence, the median ridge, which extends from the alveolar arch and terminates in the point of the anterior nasal spine.*

The premaxillary wing is posterior to the anterior nasal spine and contiguous with it. The wing projects upward from the midline of the upper face of the floor of the pyriform aperture.

* Embryologically the point of the anterior nasal spine is derived from the two premaxillary bones. The median ridge and base of the spine comes from the two maxillary bones.

The cartilaginous portion is formed by the cartilage of the septum that is attached inferiorly to the anterior nasal spine, to the premaxillary wing, and to the anterior part of the vomer.

The membranous septum is made up by the juxtaposition of two mucocutaneous flaps. The layers of the membranous sep-

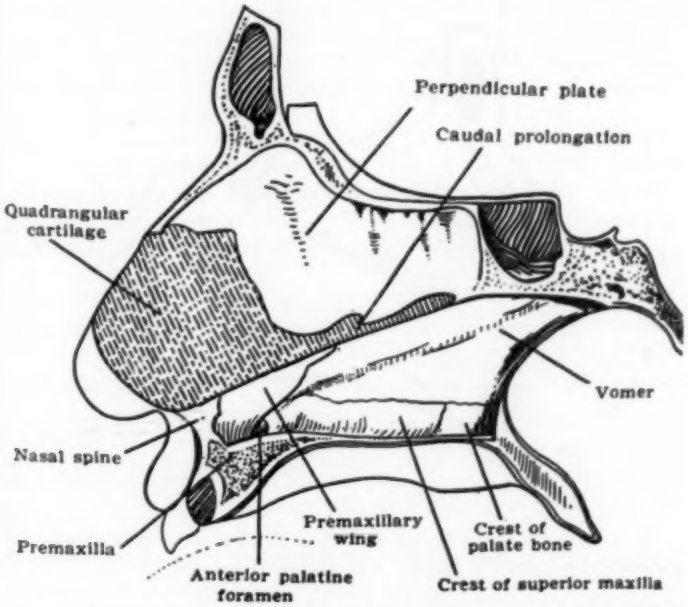


Fig. 1. The Antero-Caudal End of the Nasal Septum (modified from Mosher[4]).

tum extend forward from the free anterior margin of the septal cartilage to the cutaneous coverings of the medial crurae of the lower lateral cartilages, which form the cartilaginous support of the columella.

2. The Adult Septum: Anatomical terms that name the structures in the various diagrams are used interchangeably.

a. Bony Portion: The premaxillary wing (Mosher[4]) has the same meaning as the incisive crest (Freer[5]). The terms crista nasalis maxillae (Loeb[3]), crest of superior maxilla (Mosher[4]), and palatal process of superior maxilla (Lothrop[6]) are synonymous.

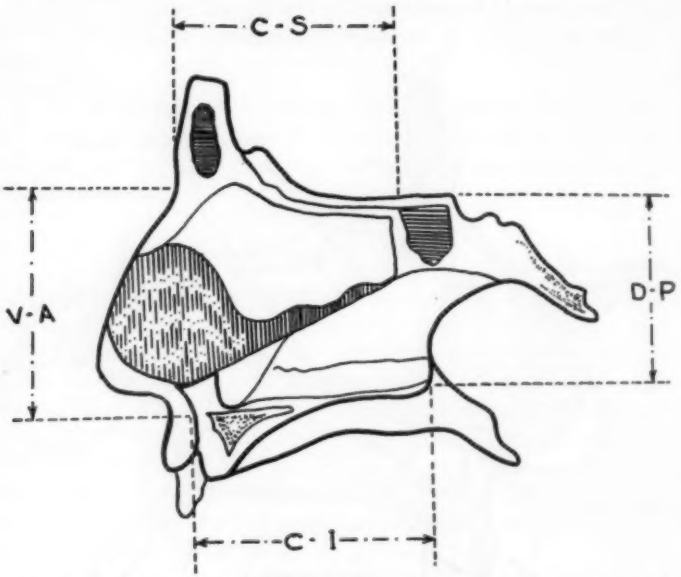


Fig. 2. Relationship of the Antero-Caudal Portion of the Nasal Septum in Terms of Position and Direction; C-S—Cephalic or superior; D-P—Dorsal or posterior; C-I—Caudal or inferior; V-A—Ventral or anterior.

b. Cartilaginous Portion: The terms quadrilateral cartilage, quadrangular cartilage (Mosher[4]), and cartilage septi nasi (Loeb[3]) are synonymous.

c. Membranous Septum: The membranous septum (Loeb,[3] Converse[7]) has the same meaning as the septum mobile nasi (Loeb[3]).

3. Terms of Position and Direction (see Fig. 2): The three fundamental planes of the body are the sagittal, the trans-

verse, and the frontal. The vertical plane through the longitudinal axis of the trunk, dividing the body into right and left halves is the median or midsagittal plane, and any plane parallel to this is a sagittal plane. Any vertical plane at right angles to a sagittal plane, and dividing the body into front and rear portions, is a frontal (coronal) plane. A plane across the body at right angles to sagittal and coronal planes is a transverse or horizontal plane.

Terms pertaining to the front of the body are anterior or ventral; to the rear, posterior or dorsal; upper is designated as superior or cephalic; and lower as inferior or caudal. The term medial means nearer the midsagittal plane, and lateral, further from that plane.

It should also be noted that the terms ventral, dorsal cephalic, and caudal are independent of the body posture, and, therefore, apply equally well to corresponding surfaces of vertebrates in general with horizontal body axis.[8]

The anterior free end of the septum (quadrilateral cartilage) refers to the free margin of this cartilage in continuity with the membranous septum. The caudal bony portion of the nasal septum refers to the area of the premaxilla.

In this thesis, the parts of the premaxilla pertinent to the caudal border of the nasal septum are the anterior nasal spine and premaxillary wings. The remainder of the premaxilla, as described by Ashley-Montagu,[9] enters into the structure of the pyriform aperture and the anterior hard palate.

4. Nares: The nares is bounded below by an internal and external naris between which lies the floor of the nasal vestibule. The inferior part of the internal narial fold extends along the crest of the pyriform aperture.[7]

5. Pyriform Aperture: This aperture is the pear-shaped anterior external opening in the bony skeleton that bounds both nasal fossae. Its lateral margin is called the ascending or lateral edge. The floor of the pyriform aperture varies with age and with the ethnic origin of the individual.[10] In some races the adult floor of the pyriform aperture has a well de-

fined edge anteriorly; the crest of the pyriform aperture. This crest is noticeably deficient in the negro race and, therefore, the floor of the pyriform aperture is continuous with and on the same horizontal plane as the floor of the nose.

REVIEW OF LITERATURE.

a. Historical: The voluminous literature relevant to the nasal septum was reviewed, and a certain pattern of thought concerning the submucous resection was noted in each decade. One of the earliest procedures consisted essentially of cross hatching, crushing, and sawing the deviated cartilage and bone.[11] Reports at the turn of the Twentieth century dealt largely with septal operations performed through an oral approach.[12] and rebuttals by proponents of the nasal approach.[14,15] Then, because of the writings of such pioneers in septal surgery as Halle,[14] Killian,[15] Freer,[5,16,17] and Ballenger,[18] the intranasal technique of performing a submucous resection was placed on a firm footing. These authors also described their individual procedures for correcting septal spurs and deformities.

The dangers of saddling of the dorsum and retraction of the columella as a sequel to partial or total submucous resection created a period of discussion.[19,20] The problem of whether to perform septal surgery on children appeared intermittently in the literature.[12,14,21] Operating techniques for correcting caudal deflections of the nasal septum received much attention.[22,23,24,25,26,27,28,29,30,31,32] More recently papers have dealt largely with operative indications and the relationship of septal deformities to the broader field of rhinoplasty.[33,34]

Many articles pertaining to the etiology of septal deviations span the breadth of the literature.[35,36,37,38,39,40,41,42]

Developmental abnormalities, trauma in childhood or later life and delayed eruption of deciduous teeth have all been stated to be factors as the ultimate cause of deviations of the nasal septum.

In 1931 Neumann[43] discussed the mucoperichondrial and mucoperiosteal coverings of the nasal septum. He noted that,

prior to that time, only Ballenger's textbook[44] contained diagrams of this anatomy. Lothrop[6] and Freer[16] (see Fig. 3), and Aymard[45] also described these soft tissue reflections over the underlying rigid structures.

As late as 1858, after more than 200 studies had been devoted to the premaxillary bone in man and mammals; its existence as a normal structure in the skull of man was denied.[46] In 1869, however, a detailed developmental account of the premaxilla in man was published.[47] From this time on, there was no longer any doubt concerning the existence of the premaxilla in man. The embryological development of the premaxillary bones in the human is described comprehensively in two papers.[9,48]

b. Development of the Premaxilla in the Embryo: In man, the premaxillae are not observed at birth, because a delicate plate of the maxillary bones extends over them. It is for this reason that the very existence of a premaxilla (intermaxillary bone) in the human skeleton was a most controversial subject. The premaxilla is ossified from two centers placed side by side. The main center arises at the middle of the seventh week and the infravomerine center at the end of the tenth week.[48]

One of the more recent and excellent descriptions of the embryology of the premaxillae is that written by Ashley-Montagu.[9] The premaxillary structures contribute to the nasal septum, as the following quotation from this paper indicates:

"With the commencement of the ossificatory process, a number of premaxillary structures make their appearance and in the following order: *1.* A keel for the reception of the anterior-inferior portion of the vomer; *2.* the anterior floor of the nasal fossa; *3.* the anterior nasal spine; *4.* the ascending processes which form the walls and lateral borders of the pyriform aperture endofacially; and *5.* the palatine processes forming the anterior part of the palate, the median septum and the anterior and lateral walls of the incisive or nasopalatine foramen" (see Fig. 4).

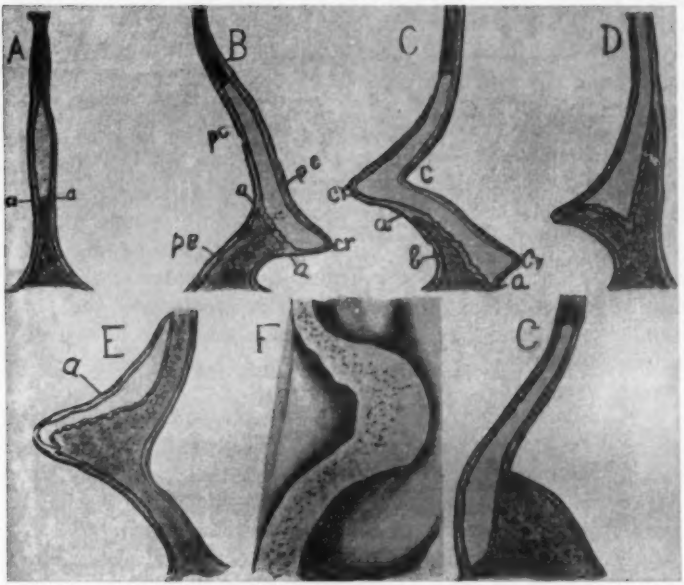


Fig. 3. A—Normal septum in vertical section showing perpendicular plate, cartilage and vomer; a-a—vomero-cartilaginous articulation showing crossing of blended periosteum and perichondrium from naris to naris.

B—Crest deflection in vertical section. a-a—displaced vomero-cartilaginous articulation slanting into naris of convexity; cr—cartilaginous crest; pc—perichondrium; pe—periosteum blending and crossing from naris to naris in the plane of the articulation a-a.

C—Type of crest deflection with slanting of the vomero-cartilaginous articulation into the naris of the concavity, with the formation of two crests and angular bending of the cartilage on itself to form a knee; c—concavity of the deflection; cr-cr—the two cartilaginous crests; a-a—line of vomero-cartilaginous articulation with crossing of blended periosteum and perichondrium; b—vomer.

D—Unusual case where flanges of vomer were wedged open by the downward pressure of the growing cartilage to form a crotch.

E—The junction of the vomer and perpendicular plate to form the bony V; a—processus sphenoidalis septi cartilaginei (caudal prolongation of Mosher), escaped from laterally opened joint and applied to side of vomer and perpendicular plate.

F—A very massive bony V.

G—Type of crest deflection without overhang of vomer, producing a nearly straight septum in the narrowed naris with typical concavity in the other. Dotted line shows where the thick, wedge-shaped vomer would ordinarily overarch the nasal floor. Such septa seem not to be in the median line, but really are so.

Mucoperichondrial and Mucoperiosteal Reflections of the Nasal Septum. (from **Freer**).

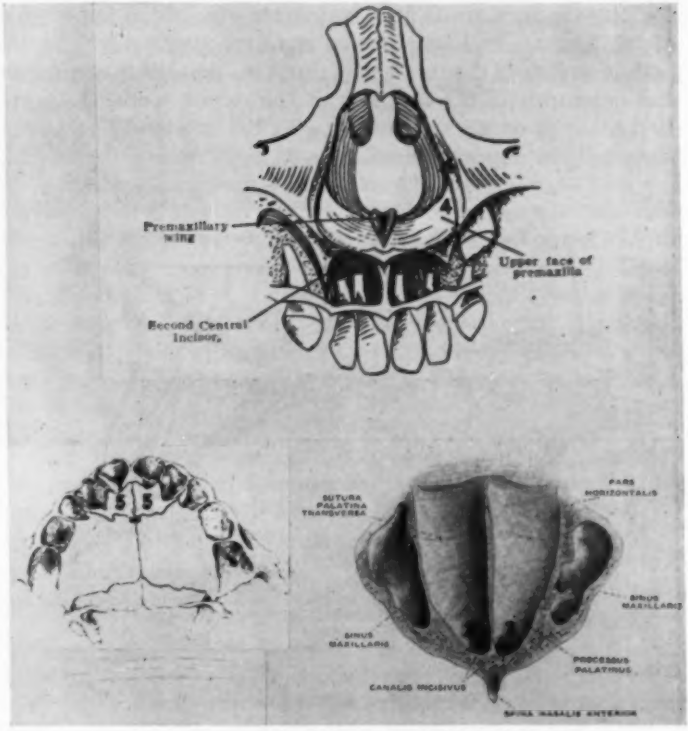


Fig. 4. Premaxillary Structures. 1—Premaxillary wing. Note the groove into which the vomer fits (modified from Mosher). 2—Anterior floor of the nasal fossa (modified from Loeb). 3—Anterior nasal spine (from Loeb). 4—Walls and lateral borders of the pyriform aperture endofacially (modified from Mosher). 5—Palatine processes (modified from Ashley-Montagu).

c. Development of the Premaxilla from Birth to Adulthood: This subject is very well discussed and illustrated in Mosher's publications[4,36,37] and is pertinent to the anatomical description of the premaxillary area.

At birth there are two structures of the premaxillary bones that become component parts of the nasal septum, namely, the anterior nasal spine and the premaxillary wings.

The nasal spine at birth is a pointed process that projects

horizontally forward from the anterior edge of the upper face of the premaxilla (floor or the pyriform aperture). The superior surface of this is slightly concave. As the antra enlarge and descend with the eruption of the second tooth, the anterior nasal spine grows upward with the crests of the palate bone and the superior maxilla.

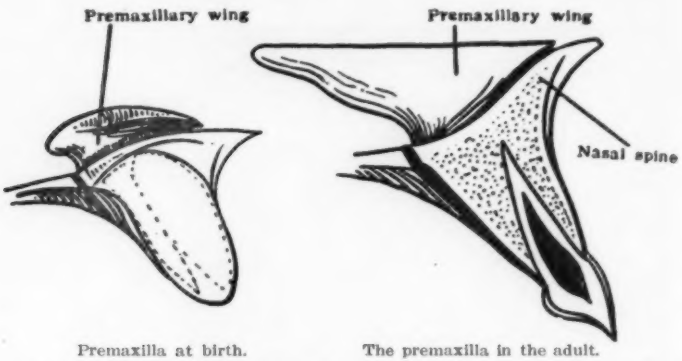


Fig. 5. The Premaxilla at birth and in the adult; note the increase in size and forward projection of the anterior nasal spine (from Mosher).

At birth the premaxillary wing projects obliquely upward and outward in the midline of the upper face of the premaxilla and is posteriorly contiguous to the anterior nasal spine. The premaxillary wings increase but little in size from birth to six years of age. From this time on they enlarge rapidly and are said to fuse with the tip of the vomer at 15 years of age (see Fig. 5).

The superior face of the premaxilla is flat like a plateau at birth and the premaxillary wing is firmly united to it by a slender neck and apparently is a part of it. In the process of maturing, the flat plateau disappears, and the superior face of the premaxilla slopes downward and backward at an angle of 45°, converting it into the shape of a moat.

In the region of the premaxillary wings the quadrilateral cartilage is thin and has its chief center of growth. The in-

fant septum is cartilaginous except for the vomer and the premaxilla with its processes. The ossification time of the various parts of the nasal septum is well described by Spielberg.[42]

When fully developed, the vomer consists of two leaves of thin bone which are united below but are open and flaring above to form a V, or gutter. The premaxillary wings arise from the posterior half of the upper face of the premaxilla and duplicate the gutter formation of the vomer. The tip of the vomer rests in the wings. The nasal spines arise from the anterior part of the upper face of the premaxilla and likewise make a slight channel into which fits the tip of the premaxillary wings. To quote Mosher's[4] summary of the gutter formation:

"The tip of the vomer rests in the gutter of the premaxillary wings, and the tip of the premaxillary wings rests in the gutter of the nasal spines, like the arrangement of the sections of the old-fashioned V shaped wooden drain" (see Fig. 6).

d. Mucoperiosteal and Mucoperichondrial Coverings of the Nasal Septum: In an original article concerning the mucous membrane reflections of the nasal septum, Neumann[43] published the first illustration of a histological section of this structure. He noted that most perforations following septal surgery occurred along the upper border of the vomer and occurred more frequently in the anterior portion than in the posterior. Another site where perforations had a tendency to take place was in the region of the crest and spine. If this complication of the submucous resection occurred more frequently in these areas, he reasoned, it must be due to the anatomical structures, and, therefore, he investigated the mucous membrane reflections of the nasal septum.

The membranous reflections surround three important sutures in the nasal septum: *1.* the suture between the quadrilateral cartilage and perpendicular plate of the ethmoid; *2.* the suture between the quadrilateral cartilage and vomer; *3.* the suture between the vomer and perpendicular plate of the ethmoid.

The mucous membrane is easily elevated over the suture line between the quadrilateral cartilage and perpendicular plate of the ethmoid, because the mucoperichondrium and mucoperiosteum are almost continuous except for a few loose fibers at the suture line.

The second suture line between the quadrilateral cartilage and the vomer presents a more intricate anatomical situation.

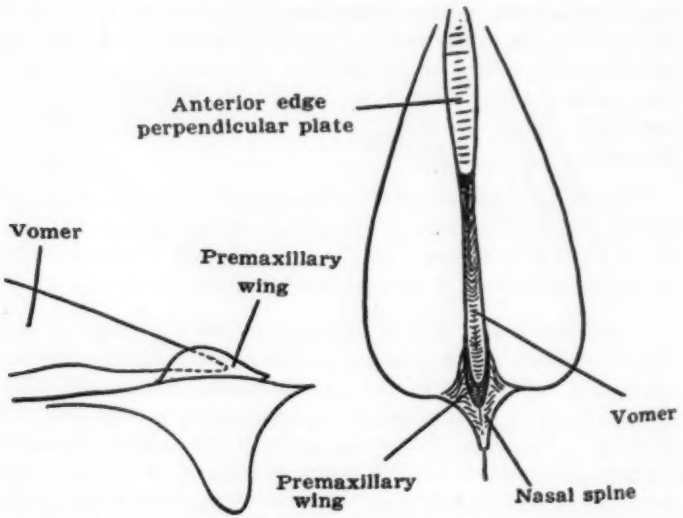


Fig. 6. The gutter formation; note the grooves and the arrangement of the anterior tips of the vomer, wing, and spine (from Mosher).

Here, the perichondrium overlaying the quadrilateral cartilage divides into an inner and outer layer. The outer layer is continuous with the outer layer of the mucoperiosteum over the vomer, but the inner one merges with the inner layer of the vomer periosteum and extends into and through the suture. This suture is wide and the inner layers which pass into the suture are thick.[43]

The suture line between the vomer and perpendicular plate of the ethmoid is quite narrow. Although the periosteum cov-

ering the perpendicular plate also divides into inner and outer layers, fewer fibers of the inner layer pass into the narrow suture line. This suture line is obliterated around 15 years of age, thereby thinning out the inner layer of fibers.[42]

Although Neumann did not make a histological study of crest formations, he thought the results would be the same as the normal septum. Killian[15] states:

"The front end of the vomer and the crest are enveloped in their own periosteum, which, therefore, covers their upper borders after the cartilage has been resected from it."

The rigid structures underneath their soft tissue coverings are visualized with difficulty, because no boundary is visible where the soft tissues are reflected onto the bone. The edge of the bone cannot be seen but must be felt for with an instrument.[5]

MATERIALS AND METHODS.

Detailed anatomic studies were made on adult cadaver heads. Many patients, ages 20 to 40, were examined during septal operations performed by the author and other surgeons.

Six cadaver heads were allowed to soften in glycerin over a period of about one month. Two of the heads were sectioned sagittally just lateral to the midline. The remaining four heads were kept intact and dissected from the frontal aspect.

In one of the dissections each important anatomical part was covered with a different color in an effort to provide more contrast when photographing the structures and their anatomical relationships.*

* A Primar Reflex camera with a F-3.5 Tessar lens was used in taking the photographs. Lighting was obtained by means of a strobe light unit that fired through a close-up ring attachment. Metal extension tubes varying from 10 mm to 60 mm. in length were used for close-up views. The most pertinent details recorded on the 2¼ x 2¼ inch size positive transparency could be cropped to the 35 mm. size. It was possible therefore, to obtain a telephoto effect without losing depth of field necessitated by the F15.6-F-8 exposure aperture of a 105 mm. lens. A polaroid filter was found to be necessary for reducing glare from the metal instruments and moist surfaces.

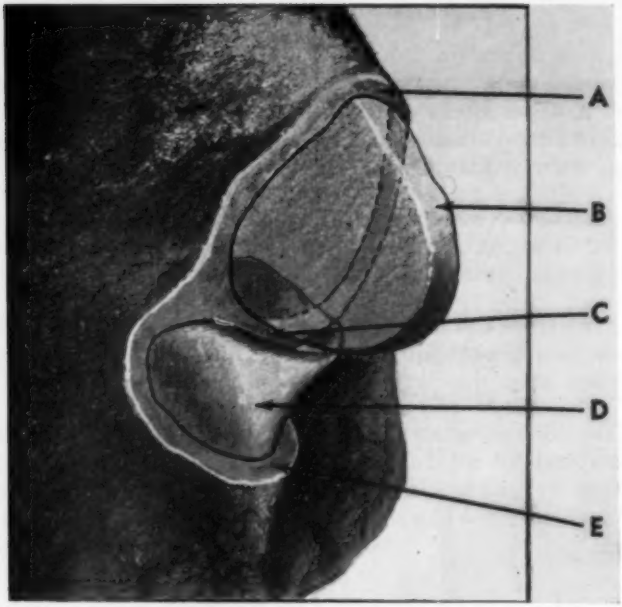


Fig. 7a. Perichondrial and Periosteal Envelopes of the Quadrilateral Cartilage and Anterior Nasal Spine (oblique view); note that the perichondrial and periosteal envelopes completely enclose their respective structures and fuse between their contact area.

A—Perichondrium; B—Quadrilateral cartilage; C—Fusion of perichondrium and periosteum; D—Anterior nasal spine; E—Periosteum.

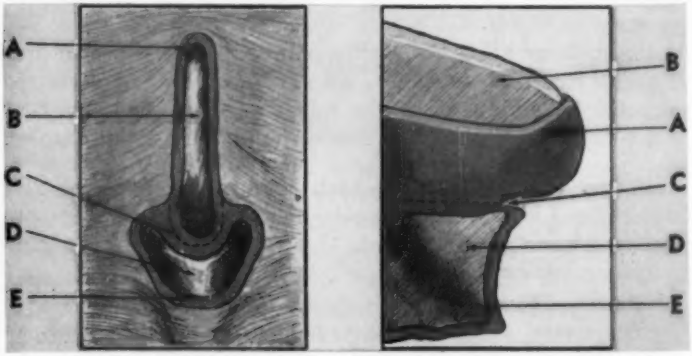


Fig. 7b and 7c. Perichondrial and Periosteal Envelopes of the Quadrilateral Cartilage and Anterior Nasal Spine (left drawing is a coronal view, and the right drawing is a sagittal view); note a type of joint capsule in the coronal view.

A—Perichondrium; B—Quadrilateral cartilage; C—Fusion of perichondrium and periosteum; D—Anterior nasal spine; E—Periosteum.

ANATOMICAL OBSERVATIONS ON CADAVER DISSECTIONS.

a. Description of the Adult Premaxilla: It will be noted from previous illustrations that in the adult premaxilla the anterior nasal spine is shaped like the prow of a ship. The anterior free end of the quadrilateral cartilage projects forward beyond the point of the prow. The caudal surface of this portion of the cartilage rests upon the superior face of the anterior nasal spine. In the region of contact between the quadrilateral cartilage and the upper face of the spine, the mucoperichondrium of the quadrilateral cartilage extends laterally and inferiorly to join with the periosteum of the wider anterior nasal spine. Perichondrium passes around the caudal surface of the cartilage and periosteum passes over the superior face of the spine. A type of joint capsule is formed within which side to side movement of the cartilage is possible (see Fig. 7a, b and c).

With hypertrophy of a wing, the cartilage is dislodged from its groove downward and laterally toward the floor of the nose. In this movement the cartilage carries with it its own perichondrium so that the caudal border is still encased within a perichondrial envelope. The perichondrial envelope is easily separated from the cartilage and the caudal portion of the quadrilateral cartilage may be levered upward.

In order to demonstrate the soft tissue reflections about the underlying rigid structures, the anterior prolongation of the quadrilateral cartilage is sectioned coronally at the point of the anterior nasal spine. The soft tissue coverings of the quadrilateral cartilage, premaxillary wings, and anterior nasal spine fuse with each other in this area.

The perichondrium on the medial side of the cartilage fuses with the mucoperiosteal envelope ascending upward and across the superior surface of the premaxillary wings (see Fig. 8).

The superior surface of the premaxillary wings is enclosed

within a periosteal envelope and a similar envelope of periosteum covers the upper face of the anterior nasal spine.

The periosteal envelopes around the anterior nasal spine and premaxillary wings can be demonstrated by presenting a dissection that begins with parallel longitudinal incisions through the periosteum on either side of the median ridge.

The periosteum is easily elevated from the smooth surface of the median ridge to the base of the spine and the joint capsule is exposed. The periosteum enveloping the anterior nasal spine continues over the floor of the pyriform aperture, the floor of the nose, and laterally toward the ascending edge of the pyriform aperture.

The caudal border of the quadrilateral cartilage is pried upward out of its perichondrial envelope to expose the fused perichondrium and periosteum covering the medial surface of the premaxillary wing.

The periosteum is very adherent to the premaxillary wings, and elevation does not proceed as easily as over the anterior nasal spine. The superior surface of the premaxillary wings is roughened and notched with numerous slight depressions.

From the dissection of the adult premaxilla, which has just been described, it can be seen that there are three conjoined compartments: the anterior nasal spine with its periosteal covering, the premaxillary wing with its periosteal covering, and the antero-caudal end of the quadrilateral cartilage with its perichondrial covering.

b. Description of the Antero-Caudal End of the Nasal Septum: The antero-caudal end of the nasal septum includes the quadrilateral cartilage and part of the premaxilla. In order to demonstrate the anatomy of these structures, the following dissection was prepared: a cadaver dissection was planned so as to provide a frontal view of the entire antero-caudal end of the nasal septum.

An incision was made through the middle of the columella

and lip and carried deeply to the anterior nasal spine. As the lip was split and retracted, an exposure of the gingivo-labial fold was obtained.

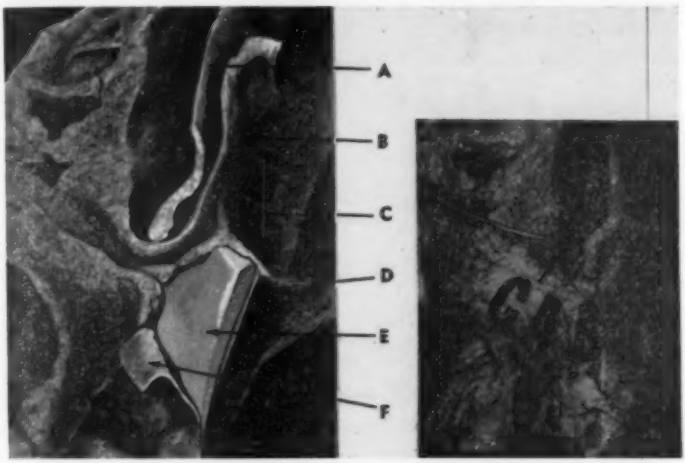


Fig. 8. Fusion of the medial perichondrium and the periosteum of the premaxillary wing when the cartilage slips downward and laterally toward the floor of the nose.

A—Cartilage (coronally sectioned); B—Medial perichondrium (note the fusion in right photograph); C—Premaxillary wing covered with periosteum; D—Alveolar edge of floor of the pyriform aperture; E—Anterior nasal spine denuded of periosteum F; G—Lateral perichondrium.

The membranous septum was divided in the midline and the dissection progressed toward the anterior free end of the quadrilateral cartilage.

An incision was made into the perichondrium surrounding the anterior free end of the cartilage in order to elevate this layer in the precise plane where it was closely contiguous to the quadrilateral cartilage. Finding this plane resulted in easy insinuation of the instrument and elevation of the mucoperichondrium from the anterior free edge of the quadrilateral

cartilage to the posterior portion of the cartilage. The attachment of the mucous membrane with the perichondrium is loose, and it is easy to separate the tissues in this plane. Separating the perichondrium in this plane may result in perforating the mucous membrane (see Fig. 9).

A study was made of the relationship of the free border of the cartilaginous septum and the premaxillary area. Arbitrarily, straight needles were inserted through the membran-

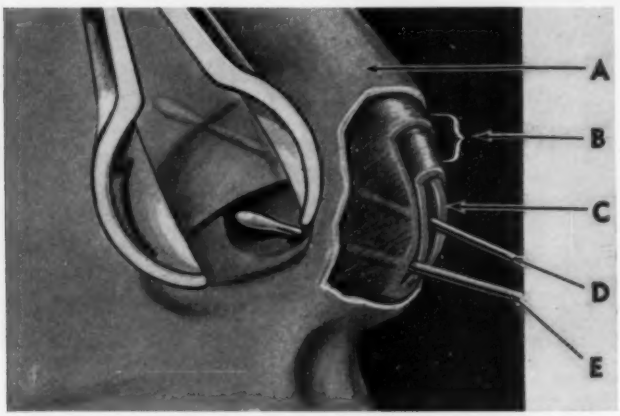
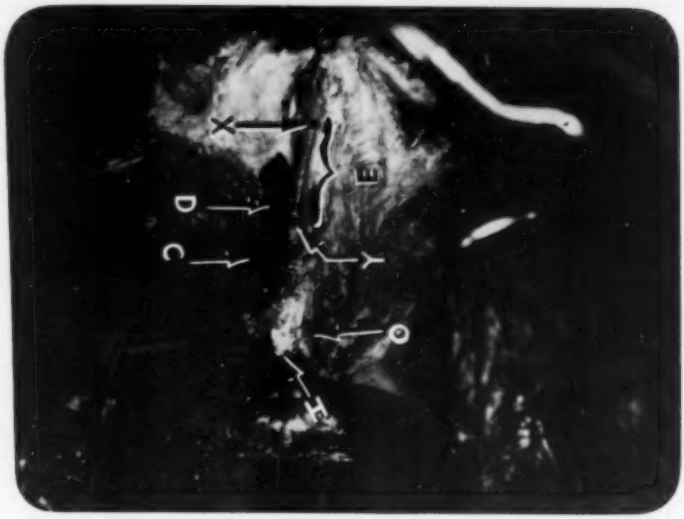


Fig. 9. Elevation of Mucoperichondrium; note the perforation when the extraperichondrial layer "E" of the mucoperichondrium is elevated.

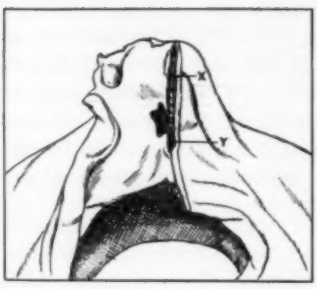
A—Skin; B—Mucoperichondrium; C—Anterior free end of quadrilateral cartilage; D—Elevator under mucoperichondrium; E—Elevator in extraperichondrial layer.

ous septum at two points. The superior point was located at the anterior free end of the quadrilateral cartilage on a horizontal level with the attachment of the upper lateral cartilage to the quadrilateral cartilage (see Fig. 10). The inferior point was chosen at the free end of the quadrilateral cartilage on a horizontal level with the superior face of the floor of the pyriform aperture (see Fig. 10).

On dissection, the needles crossing the anterior free end of the quadrilateral cartilage were designated with two colored marks (see x and y in Fig. 10a). It can readily be seen that



A.



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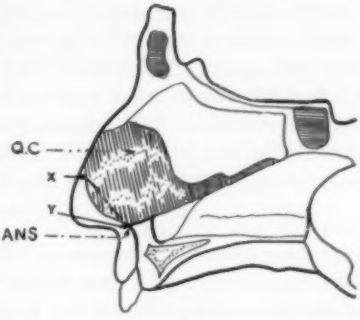

C.

Fig. 10a, b and c. Limitations of intranasal septal incision. An imaginary line connecting x and y in frontal view Fig. b; corresponds with bracketed area E in frontal view Fig. a; this also corresponds to the line x-y in lateral view Fig. c; note that the premaxillary area is caudal to points y as well as bracketed area E.

Fig. A. H. Anterior nasal spine; O. Periosteum of anterior nasal spine; C. Floor of nasal vestibule; D. Perichondrium; E. Anterior free end of qudrilateral cartilage corresponding to line x-y.

Fig. B. is a schematic line drawing of Fig. a.

Fig. C. Q.C.—Quadrilateral cartilage; ANS—Anterior nasal spine.

the antero-caudal part of the quadrilateral cartilage, the point and the median ridge are not visualized caudal to a line bracketing the two points x and y.

If x and y are connected as with any intranasal incision, it is apparent that the premaxillary area with its entire periosteal envelope is not exposed (see Figs. 10b and 10c).

SURGICAL ANATOMY.

This division of the subject is concerned with an application of the descriptive anatomy of the antero-caudal end of the nasal septum to surgery on this area. One of the frequently used surgical techniques that utilizes to advantage a knowledge of this area is the Cottle incision in the anterior free end of the nasal septum, and his procedure for undermining the base of the nose. The details for making the incision are well discussed and fully illustrated in Cottle's publications.[2,27,34]

It is important to re-emphasize the fact that the elevation of the mucoperichondrium from the quadrilateral cartilage should begin precisely in the plane where these two structures are contiguous. The bluish color of the bare cartilage should be carefully looked for before elevation of the mucoperichondrium progresses in a posterior direction. If the dissection is carried backward in a split layer of the mucoperichondrium, a perforation in the mucous membrane will result (see Fig. 9).

After the incision is made, a varying portion of the septal operation may be performed. Then, a curved, Knapp scissors is inserted into the lower edge of the incision. The back of the scissors is held firmly against the median ridge of the anterior nasal spine as the blades are opened up and soft tissues are spread in order to create a pocket. A speculum is next inserted into the pocket and placed into a position to straddle the median ridge. A longitudinal incision is next made into the soft tissue on each side of the median ridge and the periosteum elevated from the anterior nasal spine. This procedure should be limited from the point and median ridge of the nasal spine anteriorly to its base posteriorly, since ex-

tensive upraising of the periosteum would lead laterally along the floor of the pyriform aperture. With the anterior nasal spine cleaned of its soft tissue covering, the perichondrial envelope of the antero-caudal end of the quadrilateral cartilage is exposed for further dissection. The perichondrium can be elevated easily from this part of the quadrilateral cartilage and the cartilage resected.

The premaxillary wings covered with mucoperiosteum then begin to come under direct vision. As more of the ventral edge of the quadrilateral cartilage is removed in an anterior-posterior direction, the premaxillary wings are also more exposed. Frequently the caudal-ventral edge of the quadrilateral cartilage may be removed by insinuating a perichondrial elevator underneath this surface of the cartilage. By exerting a prying upward motion, the cartilage can be lifted out of its perichondrial envelope, and, thus being cleaned of its perichondrium, it may be eliminated with a biting forceps.

One can incise the mucoperiosteal sheath covering the premaxillary wing through its anterior edge and superior surface and then continue the elevation in an antero-posterior direction. This can be done, even though the periosteum is somewhat adherent to the underlying bone. After freeing the underlying rigid structures from their soft tissue coverings, the bony portion may be handled by several different methods. It may be infractured, split sagittally with a chisel, fractured and removed with a Koeffler punch, or detached from the maxilla with a 4 mm. chisel and segments removed with a forceps.

Another method of uncovering the rigid structures from their soft tissue reflections is as follows: A Pierce elevator, or similar instrument, is inserted on the concave surface between the mucous membrane flap and the perpendicular plate of the ethmoid. The instrument then is carried downward to pierce the vomerine suture fibers from above. The inner fibers are loose at the suture of the perpendicular plate of the ethmoid and the vomer, and are easily perforated. The mucous membrane is raised down to the crest of the superior maxilla. The flat surface of the Pierce elevator is held firmly against the vomerine bone and is carried forward toward the caudal end

of the nasal septum. As the instrument sweeps forward, the mucoperiosteum covering the vomer is separated easily from that underlying bone.

The elevation is more difficult at the wing, and gradually more force must be exerted in pulling the instrument forward. The fibers will suddenly give way and the elevator appears in the gutter of the anterior nasal spine. The premaxillary wings are chiseled free and splintered upward. The bony pieces are then removed from the mucoperiosteum covering their convex side by grasping a free edge of the bone with a Ferris-Smith forceps and gradually rocking them loose. The bone is taken from its covering rather than elevating the covering on the convex side.[1]

The floor of the pyrioform aperture is quite wide and this fact is of some importance when chiseling free the premaxillary wings. When a 4 mm. or 7 mm. sized chisel is used as the instrument of choice, caution must be exercised to avoid driving the blade into the upper face of the floor. Properly placed, the blade should clear the inner nasal edge of the pyriform floor as it is driven into the premaxillary wing; also, to prevent hemorrhage, a determined effort should be made to avoid striking the vessels coming up to the floor of the nose through the nasopalatine foramen.

DISCUSSION.

Considerable attention is given to the "gutter formation" of the premaxillary wings, vomer, and nasal spine. The various soft tissue envelopes that cover the rigid underlying structures result in three conjoined compartments: the anterior nasal spine, the premaxillary wing, and the antero-caudal end of the quadrilateral cartilage.

Certain features of the mucoperichondrial and mucoperiosteal coverings of the nasal septum indicate the possibility of connecting the conjoined anterior compartments with the posterior portion of the elevated mucous membrane flap. The main feature of the coverings seems to be the ability to penetrate the inner mucoperiosteal fibers at the vomer and per-

pendicular plate suture line from above downward toward the floor of the nose and then by coming forward on the concave side to elevate the coverings from the vomer to and into the premaxillary area.

In performing a submucous resection, it was generally agreed, at the beginning of the Twentieth century,* that better perspective was obtained with the oral approach than with the nasal method. The pioneers of the submucous resection rejected the oral approach for such reasons as poorer healing, prolonged post-operative discomfort, and longer operating time, and adopted the intranasal method.

The surgical approach to the anatomical structures of the antero-caudal portion of the nasal septum should result in a visualization of these structures that more closely resembles that obtained with the discarded oral technique.

It is obvious from a study of a frontal dissection on a cadaver head that any of the standard submucous resection incisions fail to give easy access to the entire periosteal envelopes of the premaxillary area. The two needle perforation marks, when projected from the frontal to an intranasal position and connected intranasally by an imaginary line, denoted the limitation of any standard submucous resection incision.

It would seem desirable to select a surgical technique that will allow the surgeon to expose adequately the premaxillary area through an intranasal incision. The Cottle septal incision and technique of undermining the base of the nose logically will not injure the respiratory epithelium, and will allow better exposure with simpler access to the bony portion of caudal basal spurs and deviations of the nasal septum. There is more postoperative discomfort because of the greater exposure, but the ease of surgical performance and lessened chance of encrusting perforations far outweigh this disadvantage.

SUMMARY AND CONCLUSIONS.

1. A detailed anatomical study on cadavers and patients of the mucoperichondrial and mucoperiosteal reflections of the

* See Review of Literature, page 1000.

antero-caudal end of the quadrilateral cartilage, premaxillary wings, and anterior nasal spine is presented. The anatomical information concerning the conjoined compartments of these structures would imply an individual surgical approach to each compartment; partial or total removal, and/or realignment of the rigid structures. With such exposure, continuation of the elevated posterior mucous membrane covering with the anterior conjoined compartment can be accomplished.

2. It was confirmed that the surgical technique of undermining the base of the nose gives an excellent exposure to the premaxillary area.

3. The anatomical findings agree with observations previously reported.

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SURGERY OF THE LARGE ARTERIES OF THE NECK.*†

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In the various fields of surgery, many noteworthy advances have been made during the past few decades. If we exclude the progress made in the control of bacterial infection by antibiotic substances, then no greater contribution to the practice of the surgery of trauma has been made than the development of the technical and supportive methods which make possible the successful replacement of large arteries in man.

Most of the serious consequences of wounds of violence depend to a large degree upon the kind and extent of the damage to the underlying large blood vessels. Methods for controlling the excessive loss of blood from operative or accidental wounds of large blood vessels, particularly in the neck, have interested surgeons almost from the dawn of medical history. A great variety of procedures has been suggested as a means of reducing the loss of blood from such wounds and, at the same time, maintain an adequate supply of arterial blood to the brain. The maintenance of an adequate flow of blood to the brain in such patients after trauma to the great arteries of the neck, therefore, becomes the responsibility of the surgeon. If the injured artery cannot be reconstituted by careful repair of the arterial wall, then the surgeon must be able to restore the continuity of the artery by replacing the damaged segment with a suitable graft of autogenous, homologous, or synthetic blood vessel of the proper caliber.

The problems concerning the surgery of the large arteries of the neck can be divided roughly into two main categories: first, those problems associated with injury to arteries or

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veins in wounds of violence, or unavoidable injuries incident to the surgical removal of large tumors of the neck; second, those problems associated with restoration of the arterial circulation to the brain in patients with congenital anomalies of the blood vessels of the neck, or some acquired disease of these large blood vessels.

Non-penetrating injuries to the neck may cause rupture of medium sized arteries or veins and give rise to large hematomas, with either extensive extravasation of the blood into the subcutaneous tissues of the neck, head and chest, or a pulsating hematoma which compresses vital structures in the neck. Early decompression of the fascial compartments of the neck, with or without tracheostomy, may become imperative if the accumulation of the blood in the neck continues to take place.

Penetrating injuries in the vicinity of the large arteries of the neck must always be considered as grave, even though no external bleeding is taking place at the time the patient reaches the hospital. A small soft clot may stop bleeding for a few hours after a tangential injury to the carotid arteries, but secondary hemorrhage is certain to follow sometime later. Tangential injuries to large arteries may result in exsanguinating hemorrhage without premonitory signs or symptoms, or extensive thrombosis of the important artery may take place. Tangential injuries of large arteries in the fresh state can usually be repaired without difficulty by sutures. Injuries of this type, if left untreated and if the patient recovers spontaneously, will always give rise to a false aneurysm or a pulsating hematoma. These complications are much more difficult to cure by surgical operation than is the original tangential injury to the artery. We are of the firm opinion that the surgical exploration of all penetrating wounds of the neck is the procedure of choice.

The segmental arteriospasm which frequently complicates injuries to large arteries of the neck can only be treated adequately by bathing the injured artery in 2 per cent papaverine solution after it has been exposed at operation. Novocainization of the regional sympathetic ganglia is of no value in over-

coming the cerebral ischemia or ischemia of the muscles of an extremity from this type of segmental arteriospasm. Hemiplegia usually results from the cerebral ischemia if this type of arteriospasm is not promptly relieved, and autolysis of skeletal muscle with the production of Volkmann's contracture may result in extremities.

With modern supportive methods, it is no longer necessary, and rarely desirable, to replenish the lost blood to its full extent and thus raise the systemic blood pressure to its normal level. This only encourages further hemorrhage by dislodging a soft clot at the site of injury to the large artery. After the defect in the artery has been repaired, then the re-establishment of normal blood volume is indicated.

Much of the surgery upon large arteries should be classified as elective surgery, and careful study and preparation of the patient is essential if the surgeon hopes to obtain lasting relief without serious complications from his operations. Successful arterial surgery depends upon adequate exposure of the injured arteries by properly placed incisions of reasonable length and careful dissection of the tissues with as little bleeding as possible.

PRINCIPLES GOVERNING SURGERY OF LARGE ARTERIES.

Operative procedures which are designed to re-establish the continuity of large arteries must be performed soon after the initial injury if restoration of the arterial circulation is to be accomplished.

The successful repair of a wound of any large artery is a difficult task, and the surgeon must constantly bear in mind that blood vessels carry a remarkable fluid which normally has a tendency to congeal. The formation of clots of blood within large arteries after they have been repaired results in the failure to re-establish the functional activity of the artery. Most arteries which require repair are either diseased or damaged by trauma; consequently, the problems are different from those which concern the surgeon who plans to suture a normal artery of man or some experimental animal. In addition to

the problem of intravascular thrombosis in the injured or repaired artery, the surgeon must concern himself with the adequacy of the arterial circulation beyond the point of injury to the artery. If our surgery is to be successful, we must understand thoroughly the mechanisms which control the peripheral arterial circulation.

Collateral Arterial Circulation. The advantages of knowing the behavior of the peripheral circulation in the extremity or in the brain after the main nutrient artery has been severely injured should be obvious to all of us. It is our duty to ascertain by actual tests what alterations in the efficiency of the peripheral circulation will take place if the major arterial trunk to that part is permanently and suddenly occluded during any surgical operation. When the collateral arterial circulation is active enough to keep the distal parts of the extremity in proper circulatory balance in spite of injury to or occlusion of the main artery, we believe it is inadvisable to spend the time and labor necessary to perform a successful repair of such an artery, since a single ligature on the proximal and distal ends of a completely severed artery will usually suffice to prevent further extravasation of blood into the muscles or to the outside. If temporary occlusion of the injured artery causes the extremity to remain cold and cadaveric in color, in spite of a satisfactory environmental temperature, it would be wise to postpone any surgical operation upon that artery. If, however, postponement of the operation is not possible because of the nature of the arterial injury, the surgeon must resort to every available means of restoring the continuity of the injured artery and of enhancing the collateral arterial circulation.

It is imperative to determine the patency of the communicating pathways between the vertebral arteries and the carotid arterial circulation before permanent obliteration of the internal or common carotid artery is undertaken. We advocate the use of local anesthesia when arteries of the neck are to be exposed by operation, in order that the cerebral effects following temporary occlusion of the carotid arteries may be studied.

If the patient shows no ill effects after 15 to 30 minutes of temporary occlusion of the internal carotid artery, we can be certain that the collateral arterial circulation of the brain is adequate and permanent occlusion of that artery will be tolerated without immediate complications.

The fatal cerebral ischemia which occasionally follows the ligation of the common carotid artery is the result of preexisting anatomic anomalies of the circle of Willis or ascending arterial thrombosis. We have followed the suggestion of Matas and have always applied an aluminum band when the common carotid artery is to be occluded, while the external carotid artery is always obliterated by a ligature. If the patient should show signs of cerebral ischemia within several hours after the operation, the aluminum band can be removed from the common carotid artery with complete restoration of the circulation.

Secondary Vasospasm. The sudden interruption of the flow of blood through a large artery usually is attended by marked vasospasm in the entire peripheral vascular bed of that artery; consequently, the degree of arterial insufficiency is often out of proportion to the local injury of the main artery. This secondary vasospasm must not be confused with segmental arteriospasm which is myogenic in origin. Reflex vasospasm can be relieved by the use of vasodilating drugs, such as the oral administration of alcohol, the intravenous administration of papaverine or etamon, or by the novocainization of the regional sympathetic ganglia. Occasionally after injury or repair of a large artery, the peripheral ischemia is so great that a sympathetic ganglionectomy or ramisection must be performed in order to bring about proper balance in the peripheral circulation.

Locally applied heat increases the local metabolism of the tissues but does not greatly enhance the flow of arterial blood to the distal parts; consequently, the deficiency of oxygen to the tissues becomes greater and local death of tissue frequently ensues.

OPERATIVE PROCEDURES UPON LARGE ARTERIES.

Contrary to general belief, there are still many dangers associated with the aseptic ligation of large arteries. Asepsis has greatly lessened the dangers of secondary hemorrhage and the secondary re-establishment of the lumen after ligation of a large peripheral artery; nevertheless, these dangers still must be kept in mind when large arteries of the trunk or neck are ligated or when the surgeon must deal with abnormally large and thin-walled peripheral arteries. Permanent occlusion will be more satisfactory if a wide segment of the artery is included in the ligature or the band, and also if the ligature or band is applied in such a way as to cause the least mechanical damage or fracture to the arterial wall. The process of necrosis of the artery will then take place slowly, and complete substitution of dense fibrous tissue for the necrotic tissue ultimately will take place.

Permanent Occlusion of Large Arteries. Permanent occlusion of large arteries can be accomplished by ligatures, cotton tape, metallic bands, strips of heavy fascia, or plugs of heavy fascia anchored within the lumen of the artery. It is necessary to remember that the portion of artery included in the ligature must undergo necrosis and the occlusion is made permanent by the substitution of living fibrous tissue for the necrotic tissue. For this reason, we do not advise the use of absorbable materials for permanent occlusion of large arteries, because these materials may stretch or loosen and thus subject a necrotic portion of the artery to the pressure of the blood within the artery. It is important to use some unyielding material, such as silk, metal or cotton tape.

The rate of necrosis of the arterial segment beneath the ligature varies directly with the size of the ligature and the size of the artery. A small ligature about a large vessel runs infinitely more risk of cutting through, with the result of secondary hemorrhage or restoration of the lumen of the artery, than does a large ligature or piece of cotton tape. We believe the ligature should vary in size directly with the size of the artery. When we are dealing with very large arteries, we believe that a transfixing suture of medium silk should be used

to prevent the tape from untying or being blown off the end of the artery.

It is well known that the cutting effect of ligatures on large arteries is greatly reduced if the artery is divided instead of ligated in continuity. We, therefore, advocate, whenever it is possible, the division of an artery which is to be permanently occluded by ligature. Occasions do arise when it is not advisable to divide the artery completely.

Aluminum bands also may be used for the permanent occlusion of arteries. These bands consist of strips of aluminum stock of 0.573 mm. thickness (23 Brown & Sharp's metal gauge) about 5-16 inch wide, for the occlusion of an artery the size of the carotid artery. Strips of autogenous fascia have been used to occlude arteries and pieces of cellophane have been applied about arteries to cause gradual occlusion by fibrosis. Both these methods have practical value.

The fundamental process of pressure necrosis by the ligature and the repair or replacement of this injured tissue is essentially the same in infected wounds as in clean wounds, except for the degree and rapidity with which these changes progress. The dangers of secondary hemorrhage are considerably greater after occlusion of a large artery in an infected wound. The infection hastens the dissolution of the segment of vessel beneath the ligature, frequently causes liquefaction of the blood clots, and greatly retards the process of repair. We believe that this is more reason to use unyielding ligature material for the obliteration of large arteries in infected wounds.

When a major artery and its concomitant vein must be ligated, it is important to ligate separately the main artery and the main vein. If the ligature includes both artery and vein, an abnormal communication between the artery and vein might result as the vessel walls beneath the ligature undergo the usual process of necrosis and replacement with fibrous tissue. The development of an abnormal arteriovenous communication in the neck might lead eventually to serious complications.

After extensive injury to both artery and vein by gunshot wounds or stab wounds, it is equally important that the proximal and distal ends of the injured artery and vein be isolated and ligated separately to prevent the subsequent development of an abnormal arteriovenous communication.

Suturing of Large Arteries. Since the successful repair of a wound of any large artery depends upon the prevention of secondary thrombosis at the site of suture, it is fortunate that we have available chemical substances which retard the intravascular clotting of blood. The most important of these substances is heparin. It is a natural anticoagulant which is normally present in the blood in small quantities. The prolongation of the clotting time of blood following the administration of heparin depends entirely upon the amount of the substance administered, and there is no phase when the clotting time cannot be altered by the injection of an active preparation of this substance. It is possible, therefore, to use heparin locally in the irrigating solutions at the site of the operation upon a large artery, and it also can be given parenterally to reduce the clotting power of the blood in the entire blood circulation. Heparin in doses of 50 mg. to 100 mg., given intravenously every four hours, usually prolongs the clotting time of the circulating blood to about 15 minutes. If the effect of heparin is to be neutralized at any time during or after a surgical operation, this can be done promptly by the intravenous injection of protamine sulphate.

If heparin is to be used following any surgical operation, careful attention should be paid to the hemostasis at the time of operation. It is wise to wait about four hours after the operation before therapeutic amounts of heparin are given. Careful observation of all patients receiving heparin therapy is essential and heparin should not be given to patients with known blood dyscrasias or obstructive jaundice. Heparin has proved to be a valuable adjuvant to the vascular surgeon. For prolonged reduction of the clotting power of the blood, we feel that dicumarol has many advantages over heparin, since it can be given orally once each day.

The most important cause of intravascular injury is the lodgment of a blood clot which has been carried distally from the heart, or from some larger peripheral artery, into an important secondary peripheral artery. The sudden damage to the intima of the peripheral artery provokes marked reflex vasoconstriction in the entire vascular bed. The stagnation of blood brought about by the vasoconstriction favors progressive thrombosis within the vascular system of the entire extremity. Damage to the intima takes place easily, and secondary thrombosis at this site begins within a few hours after the embolus has lodged. If there is evidence of progressive intra-arterial or intravenous thrombosis, it is imperative that we use anticoagulants to retard the clotting mechanism of the blood. The decision as to whether the acute arterial occlusion should be treated by conservative means (such as vasodilating drugs, optimum resting level, paralysis of sympathetic nerves, and passive vascular exercises), or whether surgical intervention should be resorted to without further delay, is still a matter which requires keen judgment based upon wide experience.

We believe acute occlusion of arteries of the neck should be considered as a surgical emergency, and every means of promoting an adequate exchange of blood through the collateral arterial pathways should be instituted without delay. General conservative measures, in addition to early heparinization of the patient, will result usually in satisfactory circulatory balance; nevertheless, the surgeon must be ready to perform an arteriotomy and remove the embolus from the lumen of the artery if an adequate arterial circulation to the brain cannot be brought about immediately. It also must be borne in mind that the operation of embolectomy is an extremely delicate one and, if great care is not exercised in the removal of the intravascular clot, the added damage to the intima will result in subsequent thrombosis and occlusion of the artery. The site of lodgment of the embolus must be determined accurately and the artery exposed with as little surgical trauma as possible. The operation is best done under local anesthesia with adequate exposure by properly placed skin incisions. As soon as the artery has been freed of the embolus, it should be thor-

oughly irrigated with a 1:500 solution of heparin in normal saline solution. The intima and the media of the artery should then be closed by a running suture of very fine silk. When further clotting of the blood within the artery takes place, especially in older people, the problem becomes more difficult. Heparin also should be administered under these circumstances, but it must be remembered that it will not affect thrombosis which has already taken place.

BRIDGING DEFECTS IN LARGE ARTERIES.

The practical aspect of bridging defects in large arteries, especially arteries where greater intraluminal pressure exists, is of great concern to us. To provide additional strength in such a vascular graft, surgeons naturally turned to the use of fresh or preserved segments of large arteries from other human beings, even though homologous grafts of all other living tissues have failed to retain their original function when transplanted into man. The technical, biologic, and legal problems incident to obtaining, sterilizing, storing, and implanting such homologous arterial grafts proved to be great obstacles to their universal use. Volumes of scientific reports have accumulated on this aspect of the subject alone.

It was only natural for all of us to turn again to the fundamental aspects of the problem and search for more suitable and more easily obtainable substances. Again the products of modern metallurgy, body tissues, animal secretions, and organic chemical compounds were fashioned into tubes and used to replace large arteries in animals, with varying degrees of success.

Soon thereafter the chemists began to make available, for experimental use, several varieties of plastic sheets and fibers which rival silk and metals in strength, smoothness and inertness. It was soon demonstrated that the body tissues tolerate these plastic substances even better than fibers of animal origin. Just as surgeons of a generation ago used silver tubes, magnesium tubes, and even glass tubes, so now we began to use tubes of impervious plastic materials, with or without a lining of autogenous tissue, to bridge defects in large arteries.

These, too, proved reasonably successful, and because of their smooth lining and their blood-repellent properties, they functioned as replacements for segments of large arteries of the neck or extremities, with relatively few complications. Hufnagel, Shumacker and their associates have been interested in the use of impervious plastic materials, and their studies have added much to our knowledge of the biologic reactions to these synthetic substances.

Our past experience in the field of vascular surgery convinced us that this problem is a physiologic, rather than an anatomic, one. Physiologically speaking, defects in large arteries must be bridged by autogenous tissues or by an elastic frame of some inert and pervious material through and about which the body cells can proliferate and form permanent protective layers.

Voorhees, Jaretzki and Blakemore offered a truly physiologic approach to this problem when they demonstrated that closely woven cloth of some of the synthetic fibers permitted the autogenesis of arterial coats of fibrous tissue, with the plastic cloth tube serving as the elastic layer of the new artery. This, we believe, is a fundamental contribution to the problem of arterial replacement, and our experience in the laboratory with fine meshes of various synthetic fibers has convinced us that "arteriogenesis" by this means is not only possible but may be a practical solution for some of our most perplexing vascular problems.

We believe that the remarkable ability of the plastic cloth to bring about "arteriogenesis" depends upon the physical property of being pervious as well as elastic. We have used cloth woven from various synthetic fibers; namely, Nylon, Dacron, Orlon, and Vinyon-N.

Sections of the aorta at the junction with these plastic tubes have shown that the Orlon or Dacron is very quickly covered by a firm layer of fibrous tissue which is an extension of the outer coats of the aorta. The inside lining of the plastic tube is made up of a thin layer of cells which appear to be fibroblasts. These fibroblasts are seen to extend from the outer

layers through the meshes of the plastic cloth. Within six days a well-formed layer of these fibroblasts lines the plastic tubes, and this layer is continuous with the intimal lining of the aorta above and below the graft. The blood-repellent properties of these plastic fibers could be responsible for the fact that thrombosis does not take place immediately, as it does in tubes of metal and animal products.

There is very little cellular reaction around the fibers of Orlon or Dacron, while there are occasional giant cells about the silk sutures which are used to hold the graft in place. We are now interested in using Orlon or Dacron fibers to fix the graft in place.

COMMENT.

In our work during the past two years, we have used Orlon and Vinyon-N in the form of patches of cloth and tubes made of the cloth or woven tubes of greater thickness and strength. The bleeding through the meshes of the cloth of Vinyon-N, which has been mentioned by Voorhees, Shumacker and Hufnagel, is related to the kind of weave, as well as to the physical character of the fiber itself. Orlon fiber is smoother and finer and can be woven tighter; consequently, it does not permit "bleeding through the mesh" and has, therefore, been preferred by many of us. Both Orlon and Dacron cloth are well tolerated by the body tissues, and both possess a degree of elasticity which closely approximates that of the elastic layer of the aorta. The hand-fabricated tubes make functional segments, but several investigators (Voorhees, Blakemore, Hufnagel and ourselves) believe that seamless tubes of these fibers, in various sizes, shapes, and with desired branches for renal arteries, common iliac arteries, and even smaller arteries, and which are now available commercially, are ideally suited for replacement of segments of human arteries.

The histologic studies indicate that the pervious and elastic matrix has served as a framework upon which body cells, principally fibroblasts from the periarterial or retroperitoneal or retropleural tissues, have been able to proliferate through the meshes. This gives a smooth layer of fibroblasts, which

serves as the intima, with heavy coats of fibrous tissue outside the plastic mesh to serve as protective coats of the new artery. We believe this is arteriogenesis and should be an ideal method for replacing even long segments of the aorta, since the functional layers appear to be developed from without inwardly. The new intima is not produced by ingrowth of cells from the artery at either end of the graft. This method is worthy of more serious study and further application in man.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Arrangements have been completed for the joint meeting of the North Carolina Society of Eye, Ear, Nose, and Throat, and the South Carolina Society of Ophthalmology and Otolaryngology September 17, 18, 19, 1956. Headquarters will be the George Vanderbilt Hotel, Asheville, North Carolina.

An unusually attractive program has been arranged, and a large attendance is anticipated.

Asheville, North Carolina, is in the mountains of Western North Carolina, and is a particularly beautiful spot in this season of the year.

For further information write Roderick Macdonald, M.D., Sec. and Treas., 330 East Main Street, Rock Hill, S. C.

EVOLUTION OF SURGICAL TECHNIQUE IN THE TREATMENT OF CARCINOMA OF THE LARYNX.*†

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Though this title sounds historical, we are not going to undertake a detailed review of the history of the surgical treatment of carcinoma of the larynx. A full account of the developments of the earlier years may be found in the literature.[1] The grim story of the early days of both laryngofissure and laryngectomy is interesting to read and provides the proper background for the best understanding of the more recent trends.

Endoscopic excision is the most limited form of partial laryngectomy. Its technique has not undergone any remarkable changes and probably the most important thing to say about it is to reemphasize the importance of the most careful selection of cases, according to the advice of LeJeune.[2]

"Laryngofissure"[3] (thyrotomy) is a term which should probably be restricted to the operation of simply splitting the larynx (or rather the thyroid cartilage) in order to gain access to the interior of the larynx for the purpose of removing the tumor and surrounding normal soft tissue. This operation is suitable for very early tumors of one vocal cord, but it is the general recognition of its limitations, and the consequent indications for its selection as a treatment method that has so securely established its place in the treatment of cancer of the larynx. The disrepute into which this operation fell during

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the early years of its history was due to its complete misuse in the treatment of tumors too extensive, and the dependence upon piecemeal removal of tumor tissue followed by electrocoagulation!

Better understanding of the indications for the operation and improvements in technique by Henry Butlin and Sir Felix Semon in England resulted in its reinstatement after its first loss of favor. It was practiced successfully by Sir St. Clair Thomson in England and by Chevalier Jackson and others in the United States during the early years of this century. In 1922 Chevalier Jackson,[4] recognizing the fact that lesions reaching the anterior commissure should be treated differently from those restricted to the middle third of the cord, presented his well known "anterior commissure technique," according to which the thyroid cartilage, instead of being divided in the midline with shears ("clipping technique"), was divided with a handsaw or a motor driven circular saw, in order not to cut through or crush the tumor. In a paper on laryngofissure presented before this Society by one of us[3] in 1940, as a candidate's thesis, a series of 50 operations was reported: 32 of them done by the clipping technique, and 18 by the Jackson anterior commissure technique. This latter technique was modified by Clerf[5] and subsequently by Kemler[6] and by Broyles.[7] Broyles added valuable scientific support for the concept of cartilage removal at the anterior attachment of the cords by his anatomical studies of the anterior commissure tendon.

At the Chevalier Jackson Clinic of Temple University Hospital we still do the classic laryngofissure operation in cases of cancer involving the middle third of one vocal cord, but we use a modified form of the anterior commissure operation in cases in which the lesion reaches the anterior commissure or crosses over to the opposite cord. In such cases we do a *partial laryngectomy* with removal of varying amounts of underlying cartilage, an operation perhaps similar to what has been called by Leroux-Robert[8] "anterior fronto-lateral laryngectomy." Even if the lesion does not quite reach the anterior commissure, we generally remove the anterior three-quarters

of the thyroid ala on the affected side. This procedure makes it easier to be sure of the completeness of removal of the tumor, and furthermore improves very much the prospects for voice development.

While we are speaking of conservative surgery of cancer of the larynx we should also mention the technique of partial horizontal laryngectomy, of Alonso,[9] which is suitable for limited supraglottic tumors.

Ordinarily we do partial laryngectomies under local anesthesia; but in borderline cases, where the excision must be fairly wide, and especially when we wish to make the final decision between partial and total laryngectomy on the operating table, we do first a tracheotomy under local anesthesia and insert a Woodbridge catheter through the tracheal incision. This leaves the larynx unobstructed by the intratracheal tube, permitting good inspection and palpation of the lesion and surrounding tissues; furthermore, this method affords equally satisfactory anesthesia for either wide partial or total laryngectomy.

Results of treatment by partial laryngectomy in 208 cases have recently been reported at the Fifth Pan-American Congress of Oto-Rhino-Laryngology, in San Juan[10]—88 per cent survival following primary treatment, with the salvage of eight patients who developed recurrence or metastasis, by second partial laryngectomy or other treatment, bringing the final five-year "cure" rate in this group of patients to 91 per cent.

One of the reasons it seems more important than ever to give the most careful attention to indications and techniques of conservative treatment is the fact that, as Ormerod[11] has stated, patients with cancer of the larynx are being seen and diagnosed at a much earlier stage than formerly, and hence these conservative techniques are applicable in a larger percentage of patients.

Total laryngectomy, like "laryngofissure," had a dismal and discouraging early history, with a high operative mortality and a high rate of recurrence. Gradually, however, with im-

proved technique, the operative mortality rate has fallen to almost zero. In our clinic the only operative death occurring in the past 15 years, in 255 laryngectomies, was due to cardiac arrest, in a patient with complete heart block. While, of course, the operation must be admitted to be a disabling one, rehabilitation has also improved, so that patients are able to find life worth living without a larynx.

Not only has the operative risk now been reduced to a minimum, but the period of hospitalization has likewise been greatly reduced. Not many years ago, the postoperative care of the laryngectomized patient involved a strenuous and complicated routine, requiring not only elaborate drainage which meant frequent dressings, but even wound irrigation at short intervals. Now, the use of antibiotics has eliminated all this, and wounds heal by first intention with pharyngeal fistula a rarity.

A simple one-stage excision is the usual procedure in all uncomplicated cases, whereas only a few years ago several of the leading operators advocated a two-stage operation. Whether a vertical or transverse T or U-shaped incision is used, and whether the larynx is removed from below upward or from above downward is a matter of the operator's individual preference, sometimes depending upon the location and extent of the tumor. The same may be said of the management of the hyiod bone. Some operators prefer simply to split it; others to remove the body, and still others to remove the bone in its entirety.

In a paper presented about ten years ago before the Eastern Section of this Society[12] we expressed preference for a basal anesthesia of avertin, supplemented by intravenous sodium pentothal, but for a number of years we have been using exclusively intratracheal anesthesia. Within the past year we have in a number of cases introduced the Woodbridge catheter through a tracheotomy incision in order to avoid the possibility of implanting tumor tissue in the subglottic larynx or the trachea, and, furthermore, as stated previously, to permit inspection of the interior of the larynx in the borderline cases,

where the final decision between a wide partial and a total laryngectomy is to be made on the operating table.

In the recent report already mentioned the percentage of five-year survival from primary treatment in 206 ("determinate") patients treated by total laryngectomy was 64 per cent, and the salvage of seven patients by further treatment raised this percentage to 67 per cent for the group initially treated by total laryngectomy. Combining the 208 ("determinate") patients treated initially by partial and the 206 treated initially by total laryngectomy, and taking into consideration the five-year salvage after treatment of recurrence or metastasis, the final five-year "cure" rate is 79.2 per cent for patients treated surgically. Incidentally, though this paper is concerned primarily with surgical treatment, it might be stated that during the same period 179 patients were treated by irradiation, with an initial five-year "cure" rate of 40 per cent, raised by "salvage" treatment of recurrence and metastasis to 47 per cent. Though Leroux-Robert and some others advise routine postoperative irradiation after laryngectomy, such therapy was not used in our series. The only patients who received irradiation after operation were patients who developed proven recurrence or metastasis. Of eight patients who had recurrence or metastasis after partial laryngectomy, subsequently treated by irradiation, only two survived five years, and of 27 developing recurrence or metastasis after total laryngectomy treated by irradiation only one survived five years. In recent years, surgical methods have been preferred in the treatment of recurrence and metastasis.

The most controversial point in connection with this subject at the present time concerns the management of the lymph nodes; *i.e.*, whether or not to do a simultaneous "evacuation" by *"en bloc"* dissection of the neck. If nodes are palpable, we feel that they should definitely be "evacuated"; if they are not palpable, the decision as to whether or not an "elective" or "prophylactic" neck dissection is indicated, should depend upon the location of the lesion. Since only 4 per cent of cordal tumors metastasize, and only 10 to 15 per cent of subglottic tumors do so, perhaps we are justified in awaiting the appear-

ance of nodes in these cases, but in view of the fact that about 50 per cent of supraglottic tumors metastasize, we should probably consider simultaneous "neck dissection" at least on the side of the lesion, as relatively indicated. In the case of lesions that are located anteriorly or bilaterally, it may be difficult to decide upon which side to operate, and perhaps bilateral simultaneous *"en bloc"* dissection should be considered. According to the experience of Jorge Fairbanks Barbosa[13] bilateral operation can be done without serious complications if one internal jugular vein is preserved. We have done more than 50 unilateral simultaneous *"en bloc"* dissections, but these operations have all been done within the past five years, so that it is not yet possible to give end-results of this procedure.

CONCLUSIONS.

The evolution of surgical technique in dealing with cancer of the larynx has been traced in the various modifications in the techniques of laryngofissure and other forms of partial laryngectomy as well as the techniques of total laryngectomy. The timely subject of indications for simultaneous unilateral or bilateral neck dissection is also considered. The authors' own end-results are summarized with the final percentage of 79.2 per cent five-year survivals following surgical treatment (328 out of 414 patients). Including all patients treated more than five years ago, by surgery and irradiation, the final percentage is 69 per cent (412 out of 593). The patients in whom simultaneous neck dissection was done do not figure in these statistics, since they were all operated upon within the last five years.

Finally, I should like to urge that all of us interested in carcinoma of the larynx and desirous of improving present methods and thus improving end-results, cooperate with the national and regional committees, and the International Committee for the Study of Cancer of the Larynx, in their efforts to standardize classification and nomenclature, as well as methods of reporting experience.

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SURGICAL TREATMENT OF CARCINOMA OF THE ANTERIOR COMMISSURE OF THE LARYNX.*

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Carcinoma involving the anterior commissure of the larynx or crossing over onto the anterior end of the opposite vocal cord even with unimpaired motility commonly recurs when treated by conservative surgery. By conservative surgery is meant any type of operative procedure less than laryngectomy, even though a segment of one or both thyroid alae together with portions of both vocal cords and an apparently adequate margin of surrounding normal tissue are removed. While it has been frequently stated by Clerf,[2] Schall[6] and others that lesions involving the anterior commissure are prone to recur when treated by thyrotomy, no figures could be found other than those of the indeterminate follow-up of LeJeune and Lynch[5] which were not based on five year results.

About 25 years ago the clipping operation for anterior commissure malignancy was advanced by Jackson[4] at which time he stated that if the method of subperichondrial dissection without cutting through the inner perichondrium was followed, the percentage of cures would be just as high as in cases in which the growth was limited to the central portion of the vocal cord. Satisfactory results by this type of surgical treatment should be secured when the lesion is confined to the interior of the larynx with no evidence of spread outside the larynx, and adequate normal tissue can be removed around the margins, yet when surgery more extensive than the original Jackson procedure was employed in a group of cases the recurrence rate was high. In the majority of cases of carcinoma

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involving the anterior commissure in which thyrotomy was employed, segments of the anterior portion of the thyroid cartilage on both sides were removed *en bloc* with the specimen. The internal perichondrium was not disturbed and the line of excision was through grossly normal tissue.

Broyles'[1] observations concerning the tendonous attachment of the anterior ends of the vocal cords to the thyroid cartilage and the absence of internal perichondrium in this locality, probably account for the many recurrences. On the other hand, extension into the anterior commissure may simply reflect the longer duration of the lesion and that the growth orig-

TABLE I.

CARCINOMA OF ANTERIOR COMMISSURE OF LARYNX
(5 Year Follow-up Results)

	No. of Cases		5-Year Survivals	Determinate 5-Year Survival Rate	
Thyrotomy	42		26	62%	
		75			51%
Thyrotomy and further treatment	33		12	36%	
Laryngectomy	79		50	63%	
		102			55%
Laryngectomy and neck dissection	23		6	26%	
All cases	177		94		53%

inated on one vocal cord with considerable time lapse before continuation into the anterior commissure, resulting in greater invasion of the tumor. In some instances carcinoma in the anterior commissure has extended through the cricothyroid membrane and thus into the strap muscles of the neck without demonstrable metastasis to the cervical lymph nodes.

A series of 259 cases of anterior commissure carcinoma operated upon at Jefferson Hospital on the service of Dr. Louis H. Clerf was reviewed, and the 177 cases followed five years or longer, form the basis of this report. Since 94 were alive and well the overall determinate five-year survival rate was 53 per cent (see Table I). The determinate five-year survival rate is based on the number of patients surviving five

years or longer in relation to the number of patients followed for this length of time and forms the basis for an accurate appraisal of the results of treatment.

The indeterminate cases present a serious problem, but a reasonable evaluation is obtained when cases are considered in the light of five-year follow-ups rather than on the basis of all cases treated, which include those which have not yet reached five years and those who were totally lost to follow-up. There were 38 patients who died from causes other than carcinoma of the larynx in whom at the time of death there was no demonstrable evidence of local or distant cancer. Twenty-six patients were lost to five year follow-up (three years or more after surgery) who, when last seen, showed no evidence of disease in the larynx. In the upper age group there were eight patients over the age of 70 in whom the cause of death was not determined. All of these cases must necessarily be considered as failures in spite of the fact that no malignancy was demonstrable prior to death.

The histologic diagnosis of squamous cell carcinoma was confirmed in all cases, and in only 27 cases was the excised portion of the opposite vocal cord free from microscopic cancer. There were, in addition, 52 cases not included in this statistical study, with carcinoma of one vocal cord treated by thyrotomy in which the lesion extended up to the anterior commissure but did not involve this site.

Exact classification is essential to proper understanding of the results of treatment. In all of these cases the anterior commissure was invaded by cancer. In those in which thyrotomy was performed all of the other criteria for the operation were met. There was no evidence of metastasis to the neck, and mobility was unimpaired. In the group in which laryngectomy was performed the growth was much more extensive with fixation of one side of the larynx, spread into the ventricles, ventricular bands, or epiglottis and involvement of the opposite cord, while in a few subglottic extensions were also present.

Although statistics can be interpreted to suit one's liking,

the important consideration in this group of cases is the number of recurrences in the cases treated by primary thyrotomy. Forty-four per cent of the thyrotomy group followed five years or longer had recurrence or metastasis. Some laryngologists contend that even if carcinoma recurs after thyrotomy, laryngectomy can still be done with a high survival rate. It was possible to salvage some of these at a later date by laryngectomy, but when the cases treated by primary thyrotomy and those salvaged by laryngectomy were considered together, the five-year survival rate was only 51 per cent (see Table II). To

TABLE II.

CARCINOMA OF ANTERIOR COMMISSURE OF LARYNX
(5 Year Follow-up Results)

	Cases Followed	Survival 5 Years and Over	Recurrence Metastasis	Deaths Unrelated to Cancer
Thyrotomy	75	26	33	12
Recurrence and further surgery	33	12		4
		38 (51%)		16

us recurrence means incomplete removal, and when carcinoma is found in the larynx, whether five, ten or fifteen years following the original growth, it is still a recurrence or continuation of the original growth. It is difficult to subscribe to the belief that a new cancer has developed. In looking back on the cases with recurrence, laryngectomy should have been the operation performed. Whereas 36 per cent of the group with recurrence treated later by laryngectomy lived five years or longer, when all the cases treated by thyrotomy plus those with recurrence and later treated by either laryngectomy or irradiation were considered, the five-year survival rate dropped to 46 per cent. Subsequent irradiation therapy in five patients with recurrence following thyrotomy was carried out with no five-year survivals. Additional surgical treatment to the larynx without laryngectomy was done in two of the cases after primary thyrotomy with no five-year survivals.

Even though there is no evidence of local recurrence in the larynx, when the lymph nodes become involved, it is risky to

do a block dissection of the neck for metastasis to the cervical lymph nodes without removing the original site of the lesion. While carcinoma may not be detected in the larynx, it probably exists, and unless the larynx is removed, together with the neck structures, the operation is inadequate. In the patients treated by primary thyrotomy there were six patients in whom cervical metastasis developed one to one-and-a-half years later, without evidence of local recurrence in the larynx, that were treated by radical neck dissection without removal of the larynx, and only one is alive four years following the original operation.

Considering that the five-year survival rate for all cases of carcinoma of the anterior commissure treated initially by thyrotomy is only 51 per cent, something further is needed to improve the results and bring them in line with those obtained by thyrotomy, when the lesion is limited entirely to one vocal cord, which approximates 75 per cent. A step in this direction can be taken if anterior commissure lesions are considered unsuitable for thyrotomy, and involvement of this area is used as a contraindication to this procedure. By this means it may be possible to eliminate the 44 per cent recurrence rate in those cases treated by thyrotomy, and the overall five-year survival rate should be greatly increased. At times, one is tempted to get by with conservative therapy when there is little evidence of extension to the opposite cord and the patient's desires in this matter may influence the physician's decision; however, when dealing with the life of the patient, this cannot be seriously considered, and percentage-wise the patient would have a better chance of living his normal life span after total laryngectomy.

In the group of 102 cases treated by laryngectomy the determinate five-year survival rate of those without metastasis was 63 per cent (see Table III). A much higher percentage of survivals was anticipated, but this included all cases with anterior commissure involvement regardless of the extensiveness of the lesion. While metastasis developed in 23 cases the salvage rate in these cases was only 26 per cent. The combined five-year survival rate for those patients treated by primary

laryngectomy with simultaneous or subsequent radical neck dissection was 55 per cent.

If the figures were interpreted literally the conclusion might be reached that thyrotomy was the operation of choice because of the similarity of five-year survival rates. In cases without recurrence or metastasis primary thyrotomy yielded a 62 per cent five-year survival rate as compared with 63 per cent for primary laryngectomy, while the combination of the patients originally treated by thyrotomy and those subsequently treated by laryngectomy for recurrence gave 51 per cent five-year survivals in comparison with 55 per cent when they were treated by primary laryngectomy and subsequent neck dissection for cervical metastasis. It must be taken into consideration that

TABLE III.

CARCINOMA OF ANTERIOR COMMISSURE OF LARYNX
(5 Year Follow-up Results)

	Cases Followed	Survival 5 Years and Over	Metastasis	Deaths Unrelated to Cancer
Laryngectomy	102	50	23	20
Metastasis with neck dissection	23	6		2
		56 (55%)		22

those cases treated by thyrotomy had no impairment of mobility, no extension subglottically or onto the ventricular band while those submitted to laryngectomy had fixation of one side of the larynx with frequent involvement of adjacent portions of the larynx. The extent of the lesion is a prominent factor in the outcome of the case, and there was no question in those treated by laryngectomy but that total extirpation was necessary.

No attempt has been made to compare the results of surgical treatment of carcinoma of the anterior commissure with primary irradiation therapy. The recurrences following thyrotomy which were treated by irradiation were all failures, and the patients did not survive. It has been demonstrated that cancer of the larynx can be treated successfully by irradiation

and in the report by Harris, Silverstone and Kramer[3] the five-year survival rate was exceedingly high.

Our primary objective in carcinoma of the larynx is to increase the number of five-year survival rates whether this be by surgical means or irradiation therapy. When the surgical treatment of anterior commissure carcinoma consists of laryngectomy the disability is more marked, but more patients will survive five years when they are submitted to laryngectomy rather than thyrotomy.

SUMMARY.

In a series of 259 cases of carcinoma of the anterior commissure of the larynx treated surgically 177 cases were followed five years or longer with an overall determinate five-year survival rate of 53 per cent.

Of the 75 patients treated initially by thyrotomy without recurrence or metastasis the determinate five-year survival rate was 62 per cent, but 44 per cent of the 75 cases developed recurrence or metastasis. Some of the latter were salvaged later by laryngectomy, making the overall five-year survival rate for those cases treated by primary thyrotomy and those with recurrences requiring laryngectomy later 51 per cent.

In the group of 102 cases followed five years or longer without metastasis treated by primary laryngectomy the determinate five-year survival was 63 per cent even though the lesions were so extensive as to preclude a more conservative surgical procedure. The overall five-year survival rate for all cases including those treated by primary laryngectomy and those that developed cervical metastasis necessitating secondary radical neck dissection was 55 per cent.

While the similarity of five-year survival rates between those cases treated by thyrotomy and laryngectomy might be taken as an indication of the value of thyrotomy critical evaluation reveals that the cases treated by primary thyrotomy had a 44 per cent recurrence rate, of which only 36 per cent survived five years or longer.

The five-year survival rate of carcinoma of the anterior commissure of the larynx treated surgically can be increased if laryngectomy rather than thyrotomy is used as the primary procedure, and involvement of the anterior commissure is considered an absolute contraindication to the conservative operation.

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UNIVERSITY OF ILLINOIS, COLLEGE OF MEDICINE.

The next Laryngology and Bronchoesophagology Course to be given by the University of Illinois, College of Medicine, is scheduled for the period November 5 through November 17, 1956. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois, College of Medicine, 1853 W. Polk Street, Chicago 12, Ill.

INTRACRANIAL COMPLICATIONS OF OTITIC ORIGIN.*†‡

Part I. Role of Antibiotics on Acute and Chronic Aural Disorders.

Part II. Observations on Children Under Two Years of Age Among 50,000 Autopsies at the Los Angeles County Hospital.

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The spectacle of suppurative meningitis, septic thrombosis and brain abscess is still fresh in the memory of the clinical otologists of the preantibiotic era. Too often they stood helpless by the side of a moribund patient, witnessing the harvest of the Grim Reaper after a long and losing battle. The development and use of the new chemotherapeutic agents have revolutionized the modern practice of otolaryngology.

Sufficient time has now elapsed to enable one to assess both the merits and the limitations of these chemical agents. McLay[25] stated in 1954 that the best published results on otogenous meningitis before 1922 were those of Mygind,[26] who reported a recovery rate of 28 per cent. Watson[37] achieved a recovery rate of 84.6 per cent between 1944-1947. Blohmke and Link[3] of Frankfurt in 1951 reported that, with the use of antibiotics combined with the cisternal drainage through Trautmann's triangle, the mortality rate was reduced to 12 per cent.

Before the introduction of antibiotics, brain abscess claimed

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a toll of 50 per cent to nearly 100 per cent, depending upon the skill of the surgeon and the stage of the disease.

Larroude,[21] of Lisbon, in 1950 recorded the mortality rate of brain abscess as 51.2 per cent. Natanzan[27] of Kharkov Clinic in 1951 accounted 18 cerebral abscess with 13 cures and eight cerebellar abscess with two cures, or a death rate of 43 per cent. Pennybacker[30] in 1950 stated that a death rate of brain abscess to be 29 per cent at the Radcliffe Infirmary, Oxford. Voyatcheck and Undrits,[36] in Russia, reported in 1953 the mortality rate to be 25 per cent. Ballantine and White[22] recorded in 1953 that mortality from brain abscess has been reduced at the Massachusetts General Hospital, Boston, from 80 per cent in the five-year period from 1936 through 1940, to 34 per cent from 1946 through 1950. Botterell and Drake[4] achieved a recovery rate of 86 per cent in 38 patients with localized encephalitis, brain abscess and subdural abscess, which had been treated with sulfonamides, or antibiotics, or both. Lateral sinus thrombosis has been practically wiped out.[27] Sepsis has been reduced from 25-40 per cent, to 4 per cent.[36]

Howard House,[17] in 1945, recorded that mortality from meningitis, in the Los Angeles County Hospital, was reduced from 89.4 per cent in 1935 to 14.3 per cent after sulfonamide compounds were used. The incidence of complications in the cases of acute otitis media was reduced to 20 per cent. The mortality rate of otitic complications was cut in half. Courville,[7] from the same institution, reported in 1955 that over a consecutive five-year period (1929-1933), prior to the introduction of sulfonamides and antibiotics, approximately 25 of every 1000 deaths were due to some intracranial complication of otitis media. During another five-year period (1949-1953), well within the antibiotic era, the mortality rate from the otogenous intracranial complications was reduced to 25 in 10,000 deaths, or one-tenth of the pre-antibiotic period.

It is proposed in this presentation to record the observations on the role played by the modern chemotherapeutic agents, over a 20-year period between 1935-1954 at the Los Angeles County General Hospital. Needless to say that the

greatest single factor contributing to the reduction of the infectious diseases has been the advent of these potent drugs.

There are two parts in this paper: Part I deals with statistics on the otitic morbidity and mortality before and after the introduction of these chemotherapeutic agents. Part II presents significant altered aspects of the otitic intracranial complications in the antibiotic era, based on the clinico-pathological investigations among 50,000 autopsies at this 3,500 bed institution. It points to certain pitfalls and shortcomings as related to the practice of otolaryngology.

PART I.

Table I indicates (a) the total number of the patients discharged from 1935 to 1954; (b) incidence of deaths from all causes, and (c) mortality from acute and chronic ear disorders for this same period. Table II shows incidence of (a) acute otitis media and acute mastoiditis; (b) chronic otitis media and chronic mastoiditis; (c) number of patients having simple or cortical mastoidectomies, and (d) those who had either a modified or complete radical mastoidectomy for relief of chronically draining ears and chronic mastoiditis. The comparison of the incidence of acute and chronic otitis media and their complications among both adults and children, before and after the use of antibiotics, is illuminating. The highest incidence of acute otitis media and acute mastoiditis in the preantibiotic era was in 1936. Then there were 589 patients among 62,129 admitted, or one in 105. Following the introduction of sulfa drugs and antibiotic substances, there was a dramatic decline in acute otogenous morbidity for seven years, 1937-1943. The lowest incidence was recorded in the fiscal year 1943, when there were 153 among 54,508 patients, or one in 356. Since 1944 there has been a spectacular rise in the incidence of acute otitis media. The largest number of patients with acute otitis media in a single year was in 1949, when there were 918 among 84,254 patients. This is a ratio of one to 90 patients. This high incidence of acute otitis media may possibly be attributable to a combination of several factors: *1.* An increased proportion of children under 10 years of age; 2. A relative increase in benign non-

Table I

suppurative otitis; *3*. The alteration of bacterial flora as the result of extensive use of antibiotics; *4*. To as yet undetermined causes. There has been an influx of general population, notably among children under 10 years of age who are highly susceptible to acute upper respiratory infections. The relative increase in non-suppurative otitis media may be due to the increasing incidence of minor as well as major allergic response to drugs.

As a result of extensive use of the antibiotics, many of the

Table II

more highly virulent strains of bacteria responsible for these infections have been eliminated from the picture. There remains, however, drug resistant strains of these organisms. In any event the virulence of the organisms responsible for the cases in recent years has apparently been greatly reduced, as reflected by the lessened number of surgical mastoiditis. The greatest number of simple mastoidectomies were performed in 1936, when there were 290 operations among 62,129 pa-

tients admitted that year. Since the introduction of penicillin and other antibiotic agencies, there has been a decline in the number of patients with acute mastoiditis who required a cortical operation. In 1955 there were only three among 98,147 patients upon whom a cortical mastoidectomy was performed. This is the lowest incidence of this surgical procedure in the history of the Los Angeles County Hospital. The incidence of chronic otitis media followed a similar pattern of rise and fall as in the acute cases. The number of either modified or radical mastoidectomies ran closely parallel to those of cortical mastoidectomies. Since 1946 there have been a larger number of radical mastoidectomies performed than the cortical mastoidectomies.

PART II.

Spectacular as has been the role played by antibiotics in the reduction of morbidity and mortality from infectious diseases, there has been a universal recognition of an increased incidence of undesirable features in the use of these drugs.[10,12,16,19,20,28,29,31,33,34,35] Among several factors influencing the antibacterial therapy, mention should be made of *1.* The development of antibiotic-resistant strains of micro-organisms; *2.* emergence of super infection due to alteration of normal human bacterial flora; *3.* hyper-sensitivity of the host; *4.* untoward side effects and toxicity; *5.* chronicity and recurrent infection following inadequate administration; *6.* unpredictable synergism and possible antagonism when two or several drugs are combined; finally *7.* one of the serious disadvantages in the antibiotic therapy is the increasing tendency of masking symptoms in clinical otology. A significant number of deaths attributed to intracranial complications continues, particularly among children under two years of age.[13] At the best their otoscopic examinations are often difficult. After treatment with antibiotics, the drum membrane appears gray, thickened and sometimes even retracted, due to lessened intratympanic pressure. The exudate is usually thick, viscid, and ropy, not favoring free drainage.

In this presentation the discussion is limited to this age group under two years. These were all treated at the Los

Angeles County Hospital since 1944, well after the introduction of antibiotics, and in each case the essential lesion was verified by autopsy. Excluding two children, who died from septicemia, there were 16 cases in which death resulted from intracranial complications. Each received varying quantities of antibiotics before admission. In almost every instance the initial symptoms of convulsions, high fever, and gastrointestinal disorders over-shadowed the presence of the infection in

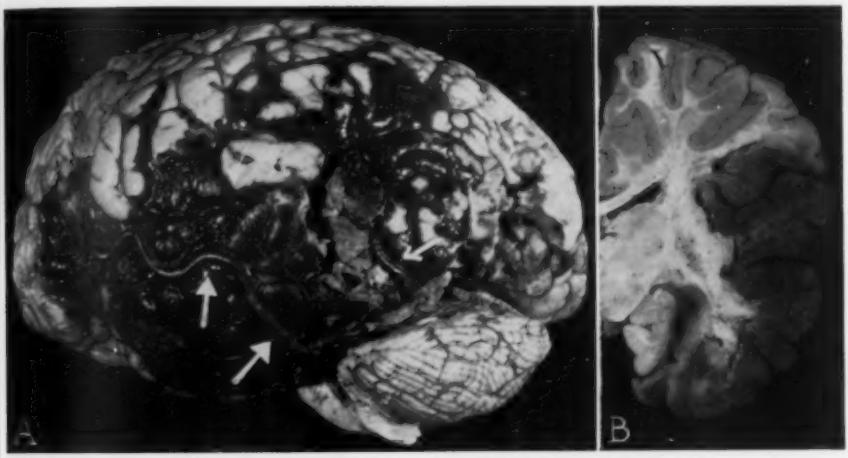

Fig. 1. Case 1. Otitic thrombosis of the inferior anastomotic and inferior cerebral vein, with subarachnoid hemorrhage and red softening of the underlying cerebral cortex.

the middle ear; thus, the acute otitis media was either unsuspected or recognized only very late in the course of the disease. The usual complications of thrombosis of the venous channels, suppurative meningitis and brain abscess in this series were found.

VENOUS COMPLICATIONS.

Two types of thrombosis of the dural sinuses are now recognized. They are *1.* septic or infectious thrombosis; *2.* marantic or primary thrombosis. To the older generation of

otologists the clinical picture of lateral sinus thrombosis and bacteremia in the presence of acute coalescent mastoiditis with recurrent chills, swinging temperature and convulsions in children, in the pre-antibiotic age, is all too familiar. Marantic thrombosis develops from alteration in the quantity and quality of the blood and from reduction in the speed of flow

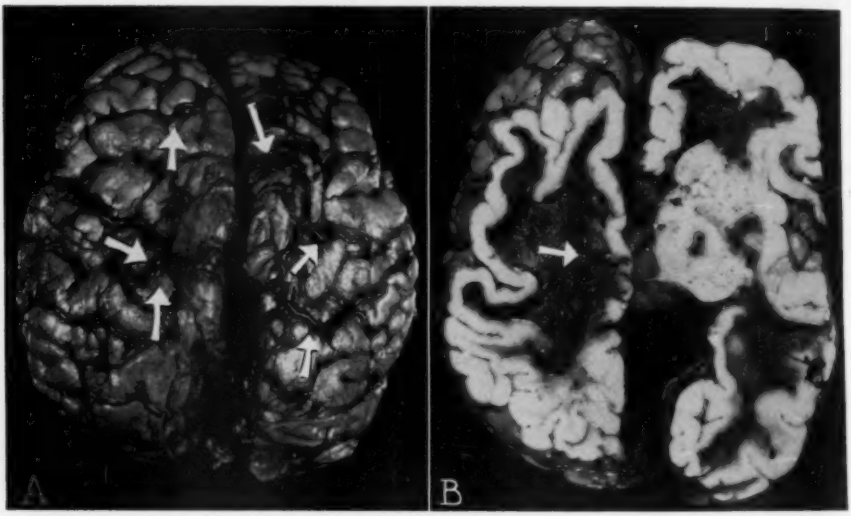

Fig. 2. Case 2. Thrombosis of the superior cerebral veins incident to otitic thrombosis of the superior longitudinal sinus. Red softening of the white centrum of both cerebral hemispheres.

secondary to dehydration, inanition and lack of normal activity. Marantic thrombosis involves predominantly the superior longitudinal sinus or straight sinus. Other sinuses are at times affected. The occlusion of the venous channels brings about edema, multiple petechial hemorrhages, softening of the basal ganglia with high mortality. In the present series there were four cases of extensive thrombotic process in the cerebral venous system. In one instance both cavernous sinuses were thrombosed.

Case 1: J. S. (L.A.C.H. No. 1057-668); a 14-months-old Mexican male was admitted with the history of upper respiratory infection and diarrhea of four days' duration. He was markedly dehydrated, acidotic and his eardrums were dull. The temperature on admission was 104°, the white blood count was 39,800 per cu. mm., with polymorphonuclears of 82 per cent. He was placed on the program of hydration, penicillin, sulfonamide and whole blood transfusion. His course, however, was downward, and he expired 48 hours after admission.

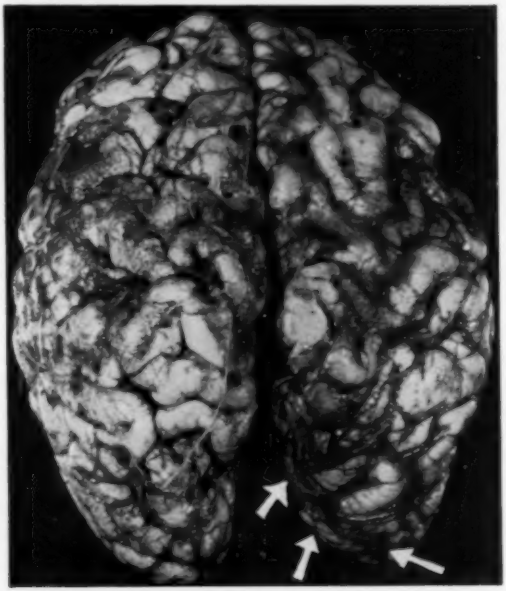

Fig. 3. Case 3. Otitic thrombosis of the right lateral and the superior longitudinal sinuses with secondary involvement of the superior cerebral veins; none evident over the posterior portion of the right cerebral hemispheres (arrows).

At necropsy examination (No. 38-429) there was found a suppurative right otitis media and mastoiditis, and an extensive venous thrombosis affecting the middle portion of the left cerebral hemisphere. There was also a softening of the left temporal and parietal lobe (see Fig. 1).

Case 2: M. T. (L.A.C.H. No. 1009-643), a well developed and well nourished Caucasian male infant, two months of age, had diarrhea and vomiting after each feeding for a period of three days. He had six watery stools on the day of admission. Both of his eardrums were injected and full. The child continued to have diarrhea with a low grade fever and mild leucocytosis. He was placed on penicillin, sulfonamide, plasma transfusion and forced hydration. His condition became gradually worse

and progressively lethargic, and he expired two weeks after admission to the hospital.

On autopsy (36953) there was found greatly distended and thrombosed superficial vessels of the meninges. There was also a complete thrombosis of the sagittal sinus, together with the superior cerebral veins, both lateral sinuses and cavernous sinuses. A mild hemorrhage had taken place over the right cerebral hemisphere and to a lesser extent over the left cerebral hemisphere. There was an extensive red softening of the white substance of both hemispheres (see Fig. 2). Staphylococcus coagulase positive was recovered from both middle ears which contained thick mucopurulent exudate.

SUPPURATIVE MENINGITIS.

Suppurative meningitis was the most frequent of the various lethal complications occurring in this series. There were ten instances of meningitis, including three cases due to pneumococcus, one each of hemophilus influenza, beta hemolytic streptococcus and meningococcus. In four instances the organism failed to grow on culture media, perhaps because their viability was severely attenuated under the influence of chemotherapy, in spite of the fact that their spinal fluid was opaque to ground glass in appearance. Infection in these cases usually develops by retrograde thrombophlebitis from the focus in the middle ear. Less frequently it may occur as an adjacent penetration from the dural layer.

There are several reasons for the failure of antibiotics in the treatment of suppurative meningitis. In the first place, the anatomical structure of the subarachnoid space permits a wide diffusion of the death-dealing organism and provides a large area for a rapid absorption of end product of the invading bacteria; second, the various antibiotic agents are incapable of rapidly penetrating the thick exudate to complete its dissolution; third, the lack of natural immunity in the debilitated and marasmic child is probably the deciding factor that tips the scale unfavorably. Histologic examination of the autopsy material showed that where blood-borne antibiotic agencies come in immediate contact with the exudate in the subarachnoid space it first becomes thin and then disappears, resulting in the establishment of bacteria-free zones. In favorable cases the dissolution of the exudate goes on progressively. Isolated patches are likewise dissolved, and the meninges become clear. In the presence of overwhelming infection, or

when the inadequate or inappropriate chemotherapy is given, death occurs after an acute or subacute course. In the first instance, a thin exudate is found at autopsy often associated with focal subarachnoid hemorrhages. To elucidate these points, the following cases are cited:

Case 8: C. B. (L.A.C.H. No. 1286-912), an eight-months-old Negro girl became irritable and feverish two days before admission to the hospital. She developed a cough and running nose. On admission, she had a tem-

Fig. 4. Case 8. Subarachnoid hemorrhage and diffuse meningitis associated with softening of the interior of the right temporal lobes (arrow) in a case of otitis media in an infant.

perature of 105° and was lethargic. Her right eardrum was injected. Diagnosis of meningitis was made. She was given penicillin, terramycin and sulfa drugs, but failed to respond. There were numerous convulsive seizures, and the child died after 18 days in the hospital and three weeks after the onset of the symptoms, from what appeared to be respiratory failure.

On autopsy (No. 46-895) the meninges were found to be somewhat opaque and the purulent exudate was most abundant over the left frontal lobe. There was a massive meningeal hemorrhage filling the basilar cisterns and almost completely enveloping the pons and medulla (see

Fig. 4). The interior of the right temporal lobe was softened. Both middle ears contained pus from which pneumococcus were isolated.

Case 10: R. M. (L.A.C.H. No. 1350-317), an acutely ill, six-months-old Mexican female infant was admitted to the hospital with a diagnosis of suppurative meningitis. At three months of age the child was treated at this hospital for diarrhea and vomiting. Four days before the present

Fig. 5. Case 10. Advanced purulent otitic meningitis, chiefly right hemispheral.

admission she had chills and vomiting. For two days the child had been coughing, with a temperature of 105°. Tympanic membranes were bulging. Myringotomy yielded purulent material from both ears, which on culture indicated the presence of a pneumococcus; the blood culture was also positive for this organism. A vigorous antibiotic regimen with supportive measure was instituted; however, she had numerous convulsions and expired the following day.

On autopsy (No. 48-173) the meninges showed marked injection. They were opaque over the dorsolateral surface of the right cerebral hemisphere, being filled with a grayish-green purulent exudate. There was a generalized edema of the brain. Histologic examination of the medulla indicated an early acute toxic nerve cell change with pyknosis (see Fig.

5). The right middle ear and mastoid contained a large amount of olive-green purulent material. The left contained only a minimum amount.

ACUTE ENCEPHALITIS AND BRAIN ABSCESS.

In pre-antibiotic days the most frequent form of otitic brain abscess was secondary to chronic otitis media and mastoiditis, often complicated with the presence of a cholesteatoma. The abscess usually develops as a consequence of the infection of the adjacent dural layer with subsequent spread either through venules or a chronic osteomyelitis. In acute otitis media and acute coalescent mastoiditis, particularly in children and adolescents, the distant lesions occur probably from a retrograde thrombophlebitis. The regional venous channels are less frequently involved. At times an abscess develops from the blood stream infection.

In the present series, there were two instances of regional encephalitis with an early central inflammation. The first case was secondary to massive thrombosis of the cerebral veins. The second one developed with recurring meningitis and unoperated petrositis. The report of these two cases follows:

Case 15: E. B. (L.A.C.H. No. 1012-831), a well-developed but poorly nourished five-weeks-old Mexican female infant, had diarrhea and vomiting for four days prior to admission to the hospital. Her temperature on admission was 105°. The pulse was 180 per minute. She weighed seven pounds. Both of her eardrums were grossly injected. A myringotomy yielded an alpha-streptococcus. She received penicillin, sulfonamides and hydration. After a stay in the hospital for a period of two weeks she expired from respiratory failure.

On autopsy (No. 36-974) there was found a bilateral thrombosis of the cerebral veins of the frontal and temporal regions, associated with regional subarachnoid hemorrhage and acute red softening of the underlying brain with secondary supperative encephalitis. Both ears showed suppuration.

Case 16: G. G. (L.A.C.H. No. 1374-591), an 18-months-old white male child was admitted to the hospital with a history of diarrhea for four or five days followed by a left otorrhea. He had finally developed signs of meningitis. Three months prior to his admission to another hospital, the child fell on his head. An electro-encephalogram there had revealed an abnormal left parieto-occipital focus. Right-sided convulsions, a left lateral rectus paralysis, a contracted left pupil, and choked disc were observed at this time. A persistent serous drainage from the left ear was noted. He was placed under heavy antibiotic therapy in that hospital. After six weeks the otorrhea ceased spontaneously, and the child appeared to be on the road to recovery. Three days before admission to the Los Angeles County Hospital the child again fell and struck his head. He began to vomit and developed a spiked temperature. Purulent otorrhea then recurred from the left ear. Despite massive antibiotic therapy and a left

myringotomy the child expired the following day. An X-ray taken two days before his death disclosed an acute left mastoiditis and petrositis.

At necropsy (No. 48-485) a heavy collection of pus was found over the tip of the left temporal lobe. The lateral ventricles were reduced in size because of cerebral edema. The left temporal lobe showed regional encephalitis with early central inflammation, as observed in the first stage of abscess formation. The clinical behavior of this child reminds the

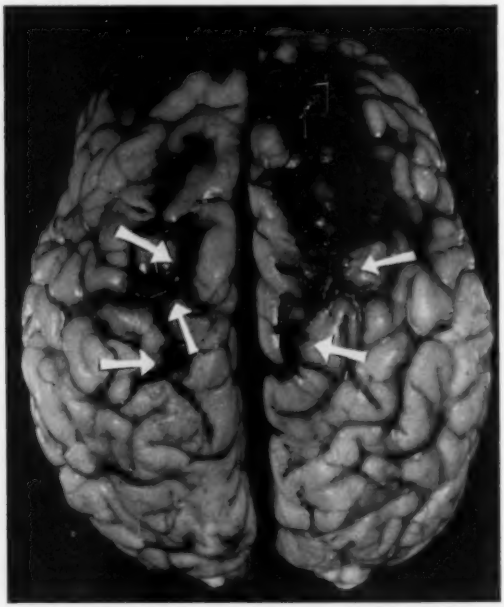

Fig. 6. Case 15. Subarachnoid hemorrhage secondary to retrograde otitic thrombosis of superficial cerebral veins (arrows).

otologist of the pre-antibiotic era, a mastoiditis due to pneumococci Type III, with its insidious onset, a slow but steady progress, a treacherous and temporary abatement of major symptoms under medical or surgical therapy, and a final dramatic explosion, resulting in a fatal issue.

GENERAL DISCUSSION.

It is of particular interest that in this series of cases the symptoms of U. R. I. were first observed in five; and an episode of initial diarrhea, vomiting and high fever had occurred

in eight patients. In three cases upper respiratory symptoms were also present in addition to diarrhea. In the past, many conflicting views have been expressed among American otologists and pediatricians[1,6,11,15,22,23,32] as to the relation of acute otitis media and gastrointestinal disorders among children. A seemingly unending controversy followed until Wishart's[38] investigation in 1930 finally appeared to settle the issue. The evidence here strongly suggests that debilitated and dehydrated children whose vitality was lowered, due to the altered electrolytes and inadequate immunity, were unable to cope successfully with subsequent or co-existing infection of their middle ears. It was recorded in a number of these instances that the symptoms abated during the administration of penicillin, but recurred when it was discontinued or given in inadequate doses. Penicillin and sulfa drugs often masked signs of grave complications.

Almost every one of these children had been first seen by general practitioner or pediatrician, and all received varying amounts of penicillin. There is a widespread notion among some practitioners of the healing art that all that is required in the treatment of infectious diseases of the ear is the administration of penicillin and other supposed-to-be-cure-all drugs. They have developed such a complacent attitude that they often omit a thorough otoscopic examination. Myringotomy has almost become a lost art.

All of these children were first admitted to the Pediatric service of the hospital. Only two of them had draining ears. Diarrhea, vomiting, dehydration, hyperpyrexia, lethargy, convulsions and signs of grave respiratory disturbances were so marked that the presence of otitis media was unsuspected in seven cases, either at the time of their admission or during their hospitalization. In four cases the tympanic membrane was recorded to be full and bulging, but no mention was made of attempts at surgical drainage. In four cases a myringotomy was performed; however, all of these 16 children at autopsy were found to have purulent exudate in one or both of their ears.

A study of this series of fatal cases of the otitic complica-

tions has brought to light some indisputable facts. Since almost every child in this series was first seen by a general practitioner or a pediatrician, the problem of prevention is one which obviously lies in their diagnostic judgment. Promiscuous use of antibiotics, often in too small quantities, can not be too strongly condemned. The choice of the drug must be based on the behavior of the bacteria as revealed by sensitivity tests. Meningitis still proves to be the chief lethal complication secondary to fulminating acute otitis media in the poorly nourished and dehydrated infant. We must all be alert in recognizing the earliest signs of meningeal irritation. To wait for a spontaneous rupture of the tympanic membrane is only to court serious intracranial complications. It is a surgical axiom that where pus occurs it must be drained. This principle remains unchanged and unchangeable even after the introduction of modern antibiotic agents.[5,6,20]

SUMMARY AND CONCLUSION.

1. Acute and chronic middle ear infections and their intracranial complications that had been observed over a 20-year period, before and after the introduction of sulfa drugs and antibiotics in a 3,500-bed general hospital, is presented.

2. For seven years (1937-1943) immediately after the institution of sulfa and antibiotic therapy, the incidence of acute otitis media was reduced from 1 in 105 to 1 in 260 patients. There has been a corresponding decrease in the number of acute surgical mastoiditis. During the past decade (1945-1954) almost as many cases of acute otitis media (one in 115) as in the pre-antibiotic era were observed. This increase was attributed to the alteration of bacterial flora coincident to antibiotic therapy, to a possible increase in number of serous otitis, and a larger proportion of children under 10 among the general population.

3. One of the serious disadvantages of antibiotic therapy appears to be the masking of symptoms, particularly among children under two years of age, often leading to fatal intracranial complications.

4. Altered aspects of the tissue response after the adminis-

tration of inadequate and inappropriate chemotherapy is presented from the pathological investigations on four cases of cerebral venous thrombosis, ten of suppurative meningitis, and of acute encephalitis among 50,000 autopsies between 1918 and 1954.

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SYMPOSIUM ON OCCUPATIONAL NOISE.

A special one-day conference Monday, September 17, 1956, will be presented by Yale University School of Medicine, Department of Surgery, Section of Otolaryngology, Department of Public Health, Section of Occupational Health and Grace-New Haven Community Hospital Hearing and Speech Center in cooperation with Liberty Mutual Insurance Company.

This one-day conference has been planned for industrial executives, physicians, safety engineers, and others who are concerned with problems of occupational noise.

Location: Fitkin Amphitheater, Yale-New Haven Medical Center, 789 Howard Avenue, New Haven, Connecticut. Date: Monday, September 17, 1956. Registration at 9:30 a. m. Fee: $15.00 per person (includes luncheon). Total enrollment will be limited in number. Only those who have registered in advance will be admitted. Further information may be obtained by writing to the Assistant Dean of Postgraduate Medical Education, Yale University School of Medicine, 333 Cedar Street, New Haven 11, Connecticut.

TONSILLECTOMY: TWO MILLENNIA OF HEMORRHAGE AND CONTROVERSY.*†

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Although the operation in point was liable to resolve itself into physical combat between the surgeon and his patient, it is one of the oldest surgical procedures. According to Celsus,[1] a Roman aristocrat who lived during the time of Christ, removal of tonsils was considered an ordinary procedure in ancient Rome. He directed that when the tonsils had become indurated after inflammations, "they ought to be disengaged all 'round by the finger, and removed; if they are not separated by this method, it will be necessary to take them up with a blunt hook, and separate them with a scalpel; then to wash the ulcer with vinegar, and anoint the wound with a styptic application." The description of this technique with such casual reference to the control of bleeding convinces me that Celsus was the first man in history about whom it could be said that he either did not tell the entire truth or did not take out tonsils himself.

One of the earliest surgeons to profess publicly his concern with hemorrhage after tonsil surgery was Aetius of Amida. Aetius, who practiced in Constantinople in the Sixth Century, A. D., compiled what is said to be the best description of diseases of the ear, nose and throat in the literature of antiquity.[2] He was afraid to take out the whole tonsil because of the danger of hemorrhage if the gland were incised too deeply; so, after pulling the tonsil forward with a hook, he cut off only the most prominent portion.

In the next century, however, Paul of Aegina[3] described complete tonsillectomy clearly and precisely, discussing in de-

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tail the prevention and treatment of post-operative bleeding. In fact, 1200 years were to pass before we find recorded again (in the 19th century) a technique as refined. Unfortunately, shortly after the time of Paul, Europe descended into the Dark Ages and, along with the general disintegration of knowledge, tonsillectomy apparently fell into disuse. When we come to the time of the School at Salerno, which was the Johns Hopkins of the Middle Ages, we find tonsil surgery limited to lancing of peritonsillar abscess.[4]

During this time the medical knowledge of the Greeks and Romans passed into the hands of the Moslems, who kept it alive until the rebirth of learning, centuries later, in Europe. Albucasis,[5] known as the most daring and brutal of the Arabian surgeons, while he removed tonsils in one way or another, greatly feared hemorrhage and did not advise the operation unless the tonsil "be of white color, and round, and the root of it, besides, small. For if the basis be very large, a flux of blood . . . is much to be feared: which has often indeed happen'd in this case, so as to prove extremely troublesome, if not dangerous."[6]

With the coming of the Renaissance, medicine on a more enlightened basis returned to Europe. Tonsillectomy, however, was slow to regain its previous acceptance and degree of development. Removal by means of the scalpel, as had been previously dscribed, was seldom undertaken because of the bleeding which was described as always violent and sometimes mortal;[7] therefore some of the more ingenious surgeons of the time tried to eradicate them by means other than cutting. The application of red hot cautery or corrosive chemicals was tried rather extensively[8,9] but needless to say did not attain much popularity with the patient. Another method involved the use of the ligature, which was developed by the great Guillemeau, whose book incidentally is a treasured collectors' item.[10,11] The tonsil was grasped and pulled out of its bed and then a noose of thread or wire was slipped around the base and tightened until the circulation was cut off. The ligature being left in place, the tonsil became gangrenous and in four or five days fell away. This method, however, was frequently accompanied by severe infection, not to speak of intense and prolonged pain.

Wiseman, surgeon to King Charles II, was brave enough to cut off the tonsil with scissors as soon as the ligature had been tightened and the blood supply stopped.

He, too, had problems, as can be seen from the following case which he described in 1686. "A Person of Honor, aged 10 years, having been some years diseased with preternatural swellings in her Tonsils . . . was brought to London . . . for Extirpation of them. . . . I placed her in a clear light, her head leaning to a bed post, Mr. Arris sitting behind holding it steady. . . . Having divided the one Tonsil, and diminished the other (by means of a long-pointed caustick stone fixed to a stick and with a Firre-stick dipt in oleo vitrioli), I caused an instrument to . . . convey a ligature about the basis of them. . . . Holding the ligature . . . with my right hand, I passed a pair of Probe-scissors . . . close by the said basis and . . . clipt . . . off the bigger part of it . . . at which instant she choaked. The Ladies seeing it, shrieked, and became troublesome . . . I desired Mr. Arris to sit still while the patient choaked. . . . She gaping I passed into her Throat the Instrument I had made the Ligature by, and raising up the Excrescence, she recovered, and cried, 'Shall I live?' I replied, 'Yes, if you sit still.' . . . But being now better informed . . . I passed my crooked probe scissors down lower, and cut it clean off by the roots, and brought it away with my Ligature. . . ."[12]

During this time it was believed that the secretions of the nose were formed in the brain and entered the nasal cavity through the cribriform plate of the ethmoid, and that the purpose of the tonsils was to absorb these secretions and send them back again. This belief goes back to the time of Hippocrates.[13] Galen,[14] called the Greek Dictator of Medicine for a thousand years, also had previously cautioned against cutting off too much of the gland for fear of injuring the voice and making the patient liable to inflammations of the lungs.

So. during this period, many physicians were opposed to removal of the tonsils for any reason, and agreed with Dionis, one of Europe's most prominent surgeons around 1700, who wrote: "Some of our ancestors propose the separation and evulsion of these glands, which operation they very easily per-

form'd, and assure us that it will never incommode the patient: I refer you to the methods which they propose of doing it, which I think very cruel; and would be glad of some better security for their success than their bare word; for the function of these glands being to separate and filtrate the secrosities which serve to moisten the tongue, larynx and esophagus, these parts must find themselves deprived of that dew, which is of great use in the tempering of the air in the lungs, and sliding the nourishment into the stomach."[15] Although in the minority, a more courageous French surgeon maintained that: "l'o'peration necessaire n'est ni dangereuse ni difficile."[16]

The era of wholesale tonsillectomy began even before the discovery of anesthesia, when in 1828 Philip Physick,[17] professor of surgery at Pennsylvania, devised the first practical tonsillotome. He reported its use in four cases wherein the operation took only a moment, causing very little pain and bleeding. This technique was eagerly taken up by his American and Continental colleagues, many of whom modified the instrument slightly and extolled theirs as more foolproof than the others, especially with regard to hemorrhage.[18] The discovery of the significance of adenoids by Meyer[19] of Copenhagen, in 1868, only added fuel to the flames. The race was now on and by the beginning of the 20th Century a tidal wave of articles on "T's and A's" was engulfing the literature. Although many of these articles contained fervent descriptions of the author's own bloodless technique, by 1921 the problem of hemorrhage was such that the entire program of the meeting of the Section on Oto-rhino-laryngology of the British Medical Association that year was devoted to a symposium of eleven papers, all on the causes, prevention and treatment of hemorrhage in connection with tonsillectomy.[20] In 1926 Jay[21] was prompted to write: ". . . it is certain that in no other operation is considerable hemorrhage viewed with so much equanimity by the surgeon."

Despite this long heritage, with consequent reluctance of many patients to have this operation performed, it is still the mainstay of the specialist's budget. It is not surprising, however, that hemorrhage can still be somewhat of a problem. In the words of Poe: "Vainly had I tried to borrow from my

books surcease of sorrow" for in the past five years, I have had eleven cases of post-operative hemorrhage following adenotonsillectomy in children. The bleeding in these cases was arterial in nature, moderately profuse, and occurred after the operation as follows: seven patients bled from the adenoidal fossa within four hours; two others bled from a tonsillar fossa also within four hours; the tenth patient bled from a tonsillar fossa two days later. The eleventh case, a physician's daughter, bled three hours following the procedure, again on the seventh postoperative day and yet again on the tenth postoperative day, each time from the right tonsillar fossa.

While representing barely 1 per cent of the total cases operated upon during this period, and although at no time was there danger of anyone bleeding to death, these eleven children caused me no little anxiety. The readministration of an anesthetic to a partially exsanguinated child whose airway is obstructed by blood can be one of the grimmest undertakings in all surgery. Even under local or no anesthesia, the customary procedures are apt to be harrowing, both to the patient and the surgeon.

The principal treatment given these children consisted of blood transfusion, this therapy successfully reducing the need for the measures ordinarily used. A surprising benefit was an apparent hemostatic effect. In all cases but two in this series the bleeding stopped before the transfusion was completed and without recourse to any other manipulation or instrumentation. As to the exceptions: it became necessary in one instance to crush a bleeding vessel in a tonsillar fossa under local anesthesia; in the other instance a postnasal plug was inserted after the child had reacted from the anesthetic.

The chief advantage of this treatment is, of course, the replacement of blood loss; however, it is often difficult to calculate accurately the amount lost. In a small child, especially, the relative blood loss is easily misjudged and on more than one occasion, prior to this series, when it seemed that a child had lost only a small amount of blood, a surprising degree of secondary anemia was later found to be present. In this series, after the bleeding had stopped, return of the pulse rate to nor-

mal was found to be a useful clinical indication that the blood had been replaced, since subsequent blood counts showed no anemia.

On the other hand, the possibility of giving too much blood with consequent reactivation of the bleeding should not be excluded. In the first excepted case the bleeding had actually stopped during the transfusion; however, the blood was continued after the calculated amount lost had been replaced. After some 200 cc. of excess blood had been given, the bleeding recurred, and it was then that the vessel was crushed. One week later this child's hemoglobin determination was a gram higher than it was preoperatively.

In using this method of treatment, consideration was given to the conservation of veins and elimination of as many venipunctures as possible. Multiple venipunctures not only deplete the limited number of readily accessible veins, but may render the child less cooperative because of added pain and terror which sometimes causes an increase in the rate of bleeding. The single venipuncture method was, therefore, used.

In this procedure, as soon as it becomes apparent that a child is bleeding postoperatively, a good arm vein is selected and an arm board is put securely in place. A new 19 or 20 gauge needle is used to withdraw the blood for cross-matching. The needle is left in the vein and a saline infusion is started through it. Preparation is made to facilitate the vomiting of blood so that the needle will not be dislodged should this occur. When the blood for transfusion is ready, it is then given through the same needle.

Strangely, this treatment is not nearly so effective in adults as in children. In the series just presented, the children ranged in age from four to ten years of age. During the same period transfusion therapy was used in the five adult patients who bled postoperatively, their ages ranging from 18 to 36 years of age. This treatment controlled the bleeding in only two cases. In the remaining three cases it was necessary to ligate a "spurter" in a tonsillar fossa.

With the exception of a mild, fleeting, generalized rash

which occurred in the case of the physician's daughter, no allergic reactions followed the use of this therapy in any age group.

SUMMARY.

1. The controversy over whether or not to take out tonsils had been going on for at least 2,000 years. The two principal factors have pertained to the danger of hemorrhage and the protective function of the tonsil.

2. The treatment of choice for moderate arterial hemorrhage following childhood adenotonsillectomy is found to be blood transfusion. This treatment exerts an apparent hemostatic effect in addition to replacing blood loss. The hemostatic effect is less in adults than in children.

EPILOGUE.

While this treatment is of value, I realize it is by no means the solution to the problem. Since all this trouble began in the early Christian era, it is only right that the nearest thing yet to the solution should also come from this time. This, the answer to an otolaryngologist's prayer, is found in Revelations, 20th Chapter, the first three verses:

> And I saw an angel come down from heaven, having the key of the bottomless pit and a great chain in his hand.
>
> And he laid hold on the dragon, that old serpent, which is the Devil, and Satan, and bound him a thousand years.
>
> And cast him into the bottomless pit, and shut him up, and set a seal upon him, that he should deceive the nations no more, till the thousand years should be fulfilled: and after that he must be loosed a little season.

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THEORY AND PRACTICE OF TYMPANOPLASTY.

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Today if we are faced with the problems of functional surgery of the middle ear in chronic otitis media, we are, thanks to modern audiology, equipped with a thorough theoretical foundation. Typical defects of the sound conducting apparatus of the middle ear create a hearing loss with a conductive component, *i.e.*, with a gap between bone conduction and air conduction in the threshold audiogram measuring from 40 to 60 dbs. If the hearing loss for air conduction markedly exceeds 60 dbs., the suspicion arises that there is a simultaneous damage to the sound perception apparatus of the cochlea present.

The various types of hearing losses are known not only from clinical experience, but have also been measured in a typical manner with the cochlear potential in animal experiments (Wever, Lawrence, Ranke, Gisselson, Wagemann, Golo).

The tympanic membrane has two functions, sound pressure transformation for the oval window and sound protection of the round window. The greatest hearing loss will be found if both labyrinthine windows are subject to sound protection; this occurs if the drum is intact but the ossicular chain interrupted (See Fig. 1). Clinically, this situation is encountered only occasionally, for example in cases with a defect of the incus after operative removal in a previous simple mastoidectomy, also in cases with atresia of the external auditory meatus and middle ear malformations. Stapes fixation does not only cancel the effect of the tympanic membrane and of the ossicular chain, but also causes a certain interference with the hy-

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drodynamics of the inner ear which will manifest itself in the Carhart notch (see Fig. 2). The total defect of the tympanic membrane eliminates to a large extent the sound pressure difference between the two windows (see Fig. 3); furthermore it produces sound reflection on the bony wall of the

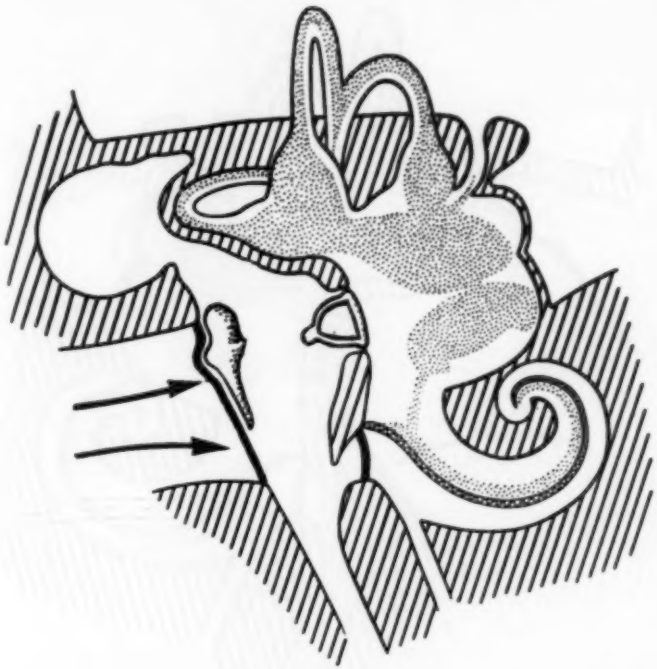

Fig. 1

promontory instead of the sound absorption which under normal circumstances occurs through the intact tympanic membrane for the main speech frequencies in a very high percentage (see Fig. 4).

Chronic otitis media causes pathological and functional defects of various degrees, sometimes of minor extent only. Adhesions frequently lead to sound protection of the round win-

dow by adhesions, or to a partial sound conduction directly to the stapes, as Zöllner has shown (see Figs. 5, 6). I must, however, emphasize that often pathological processes will improve the hearing ability, for example secretions and granulations, which close a perforation or have the effect of an

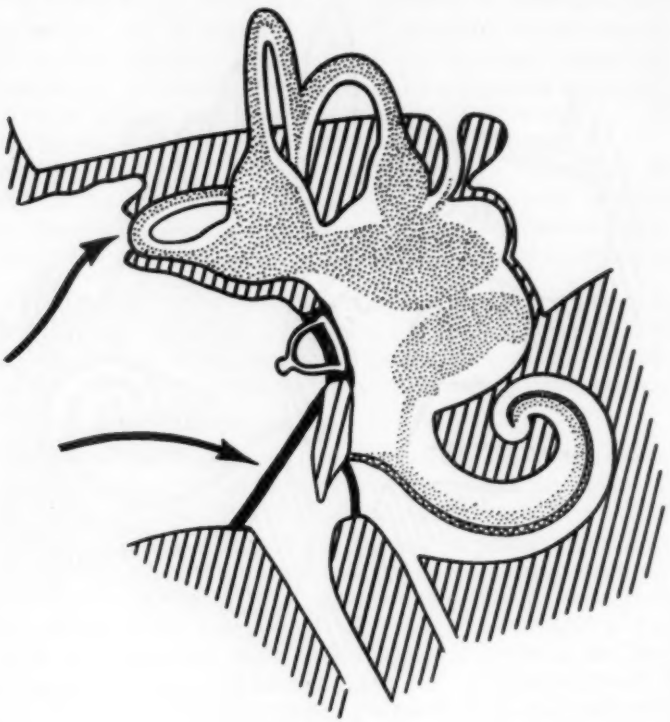

Fig. 2

artificial drum membrane, or especially masses of cholesteatoma which in a defective ossicular chain produce a certain amount of sound conduction to the oval window. The indication for tympanoplasty, therefore, is often given even with relatively good hearing and with a small conductive component only, in order to cure a pathological process or to prevent a

reinfection of a dry middle ear. Today we are interested in a definitive cure of all cases of chronic otitis media, especially those with good middle ear function so that further destruction of the middle ear, or damage to the function of the inner ear, can be prevented. It is self evident that the removal of

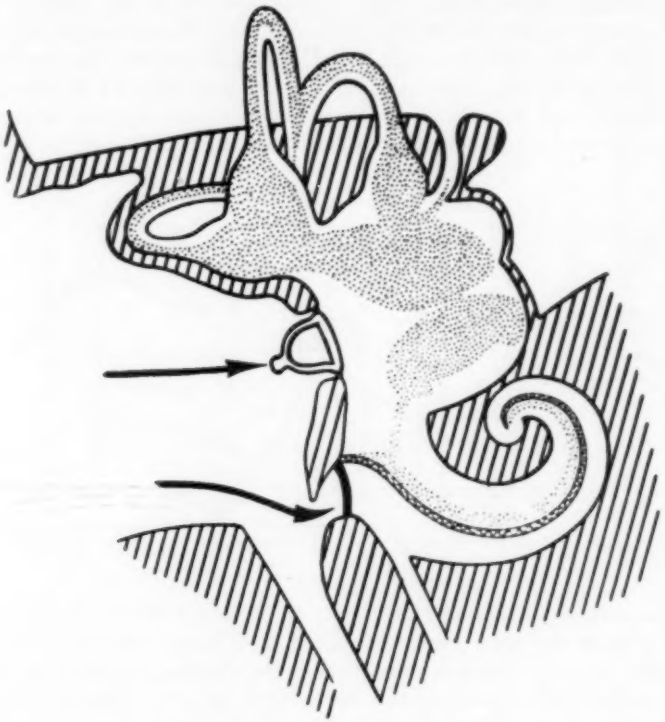

Fig. 3

the pathological changes is the first task of a tympanoplasty; thereby it might become necessary to lower the functional performance of the middle ear to the extent to which it is based on the pathological processes, before it can be rebuilt again.

Theoretically the possibility exists, that disturbances in the transmission of the acoustic stimuli through the middle ear to the organ of Corti might not be recognized. First Wever and Lawrence, later on Ranke, Keidel and Weschke, Gisselson and Richter, have shown in animal experiments that the complete blockage of both labyrinthine windows leads to a very pronounced cancellation of the hydrodynamics in the cochlea, to a very marked reduction of the cochlear microphonics. Clinically, this situation must lead to the assumption of a marked damage to the inner ear. From the experiments of Ranke, Keidel and Weschke we have, however, also learned that in order to bring about this effect the round window niche must

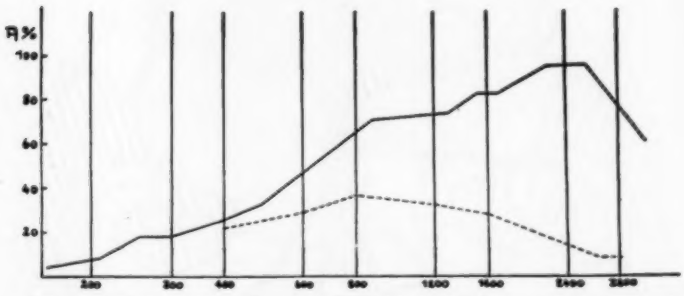

Fig. 4. Sound-absorption of the tympanic membrane ————normal; ..... complete defect of the tympanic membrane (after Menzel).

be completely closed right up to the round window membrane without any interposed air bubble. If a large cushion of air is left in the round window niche the hydrodynamics of the round window will not be interfered with. It is difficult to say whether in clinical practice interference with the hydrodynamics sometimes is actually mistaken for inner ear damage. I do not, however, believe that the question is of great practical importance. From the operative findings in more than 500 tympanoplasties I can state that a complete closure of the round window has only once been observed and that it, therefore, will be still rarer in combination with complete or incomplete closure of the oval window.

In contra-distinction to otosclerosis, where we have almost exclusively to deal with ankylosis of the stapedial foot-plate, chronic inflammatory and adhesive changes of the middle ear cause the most heterogeneous functional findings; therefore, the tympanoplasty calls for constant variations in the opera-

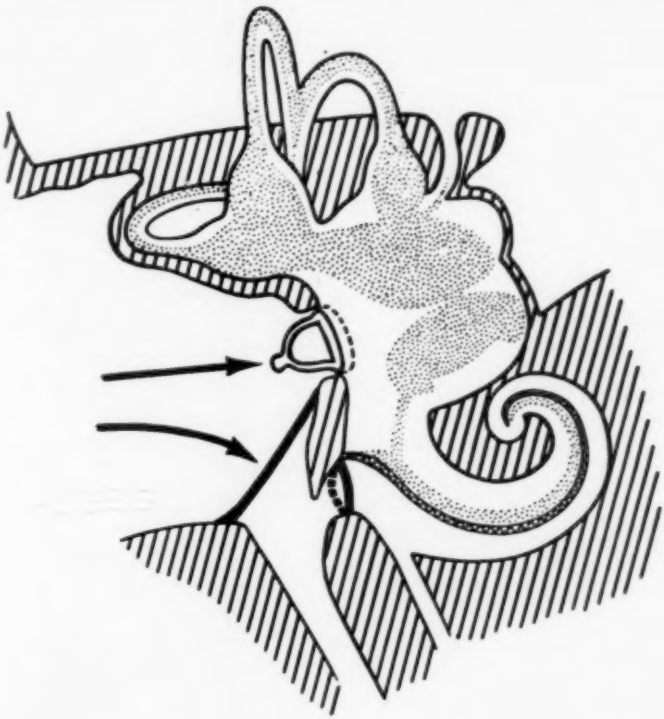

Fig. 5

tive technique. The audiological findings, however, in these operations frequently lead to certain operative ways of reconstruction of the sound conducting apparatus of the middle ears which I have, therefore, classified into five types. In principle each tympanoplasty has to be carried out according to one of these types (see Fig. 7).

Type I. Practically rebuilds a normal middle ear; a tympanic membrane with both functions, sound-protection for the round window and the scala tympani, and sound pressure transformation over the ossicular chain to the scala vestibuli.

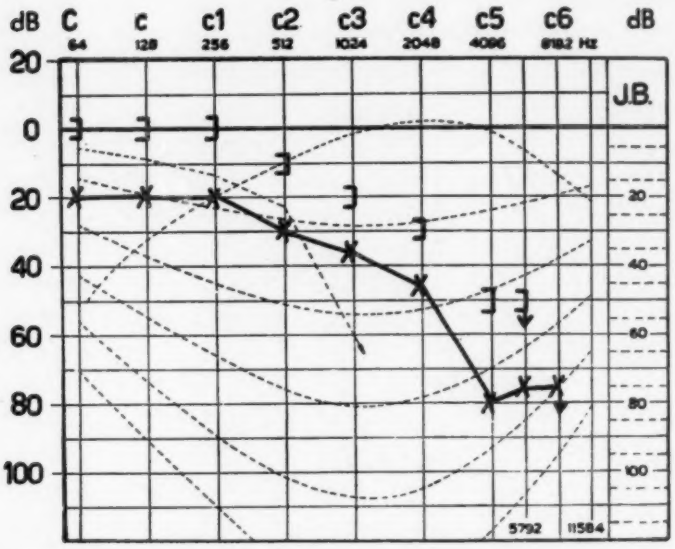

Fig. 6. Spontaneous columella-effect after frequent otitis media during infancy. Tympanic membrane transparent, mobile, on stapes adherent. Defect of the long process of the incus. Presbyakusis.

Type II. Tries to preserve, in spite of slight defects of the ossicula, a middle ear of about the normal size and depth between tympanic membrane and promontory, including the epitympanum.

Type III. In this type large defects of the malleus and incus make the removal of the ossicular chain and of the epitympanum necessary. The tympanic membrane, in order to function as sound pressure transformer, must be directly connected with the head of the stapes. The frequent spontaneous connection between tympanic membrane and the stapes through adhesions has been named "Columella-effect" by

Zöllner, "Myringostapediopescie" later by Juers. In 1901 Matte operatively achieved and published such a connection. Through a reposition of the tympanic membrane over the stapes and the lateral semicircular canal, the mesotympanum is narrowed in the promontorial region, and in this way there is an increased danger of formation of adhesions.

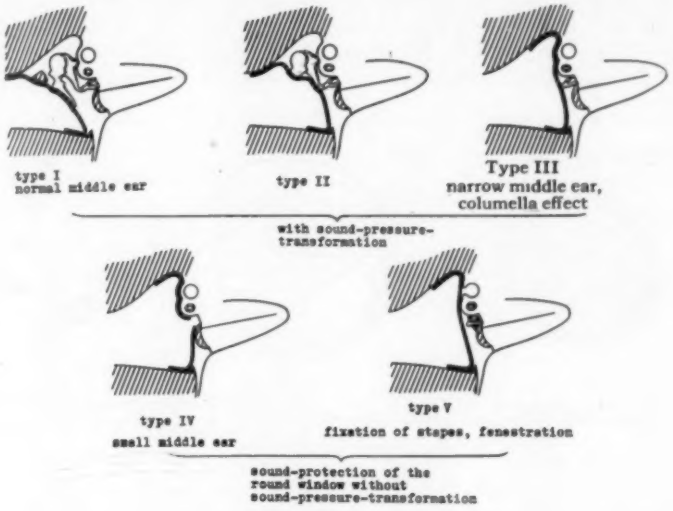

Fig. 7

Type IV. In this type the stapedial foot-plate is movable, but the crura are missing. This type must give up completely the sound pressure transformation. The oval niche is left open toward the auditory meatus. The tympanoplasty reconstructs a middle ear consisting only of the tube and the hypotympanum with sound protection for the round window. This operation was first done by Moritz.

Type V. In this type the stapedial foot-plate is fixed and a fenestra novovalis is necessary.

In theory we can predict quite accurately the results of these tympanoplasties. In Types I-V the tympanic membrane fulfills

both functions, sound protection and sound pressure transformation. If this is done in an ideal manner, no conductive loss should remain; it should be reduced to zero.

The effect of the ossicula in the transformation of the sound pressure is very small, after von Békésy it is only 1:1.3. The effect of the tympanic membrane upon the oval window is, however, 1:17, corresponding with the relationship of the sizes of the two surfaces to each other which had already been measured by Bezold, Edelman and also by Frank. We have converted these relationships into decibel figures, Güttner.

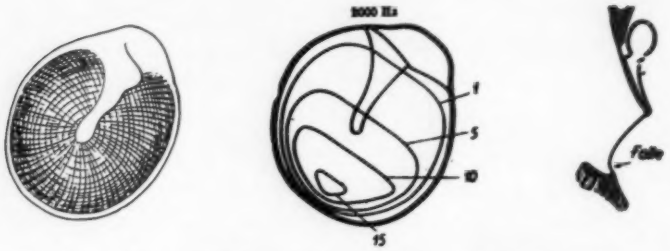

Fig. 8. Network of collagenous fibres. Curves of equivalent amplitudes of the tympanic membrane (frequencies below 2 400 Hz) (after G. v. Bekesy). Fold of the maximum of amplitude (after G.v. Bekesy).

The loss of the leverage effect of the ossicles, which occurs in Type II, and especially in Type III, amounts theoretically to 2.5 db only.

If the loss of the ossicles amounting to 1:1.3 is combined with that of the tympanic membrane amounting to 1:17, then this product amounts to 1:22, which is converted into decibel figures of 27.5 db. The loss of the entire sound pressure transformer, therefore, causes an air conduction loss of 27.5 db. This theoretical figure closely corresponds with the figures obtained from thousands of fenestrations in which one must sacrifice the sound pressure transformer for the scala vestibuli, and can only preserve the sound protection for the round window through the tympanic membrane; therefore, the typical result of a fenestration operation lies between the 25 and

30 db hearing loss for air conduction, as compared with bone conduction (see Davis and Walsh). Types IV and V, which are audiologically identical with a fenestration for otosclerosis must theoretically reach these figures.

Whether these results will actually be obtained depends to a large extent upon the quality of the new tympanic membrane. The tympanic membrane is a stiff membrane which vibrates around its lower fold, as has been shown by von Békésy. The stiffness of the membranes is due to the contained

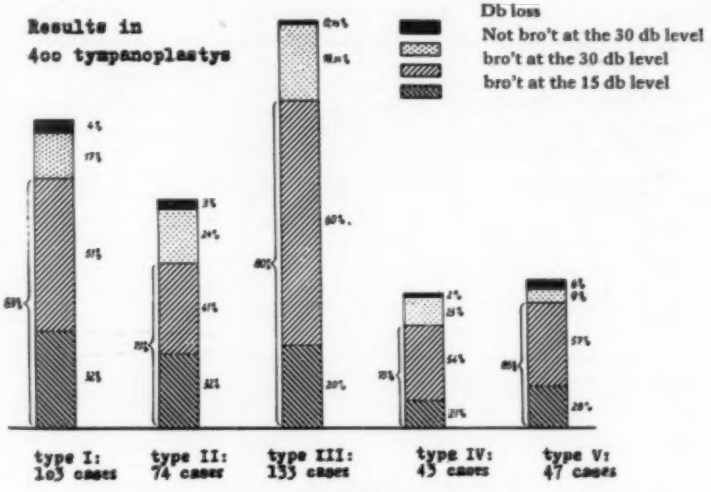

Fig. 9

collagenous fibres which, as Kobrak pointed out, cannot be stretched (see Fig. 8). If large parts of the pars tensa are well preserved in quality, the effect of the sound pressure transformation and also of the sound protection can be restored more easily. At the place of the perforation or atrophy we cannot restore the natural structure of the tympanic membrane; therefore, we must be aware of a deterioration in the quality of the new tympanic membrane as compared with that of a healthy one. If, for instance, the effect of the sound

pressure transformation of the drum membrane is reduced from 1:17 to 1:5, we get a hearing loss of 13 db. If at the same time the leverage effect of the ossicular chain amounting to 2.5 db is lost, we get altogether a conductive loss of about 15 db. The result in a tympanoplasty of Types I-III, as in stapes mobilization, with reconstruction of the sound pressure transformation is, therefore, called good as long as the conductive loss is below 15 db for the frequencies of 500 to 2000. If there is a conductive loss from 15 to 30 db for the Types I to III, I call the result only satisfactory.

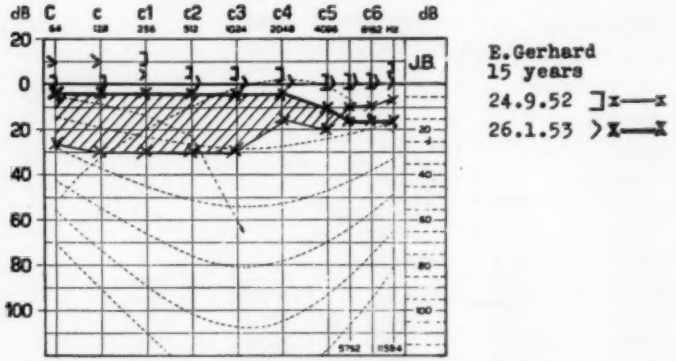

Fig. 10. Small central perforation. 30.9.51 Tympanoplasty Type 1; Ossicular chain intact. Free skin-graft, overlapping. 30.4.53 New tympanic membrane mobile.

If Types IV to V, as in fenestration, there are no such differences. Since in these types only sound protection is present, all the results can be called good which show a conductive loss of less than 30 db. All results of an audiological operation on which the conductive loss is greater than 30 db for the main area of speech, is theoretically to be regarded as unsatisfactory, even though the operation produced a definite increase in the hearing. I believe that I gave you a theoretically well founded classification of our audiological operations.

In demonstrating the results of 400 tympanoplasties, I ask that you take into consideration the fact that therein are

shown all operations, even those from the early stages of my operative endeavors. In order to study the limits of the indications and the methods of the operative technique, I have operated on all cases of chronic otitis, without exception, providing there were no contra-indication through the danger of endocranial complications. Even ears with marked and fetid suppuration, heavy granulations and extensive cholesteatoma were operated upon. Almost invariably a one-stage operation was performed; only fenestrations in Type IV were done in a two-stage procedure (see Fig. 9).

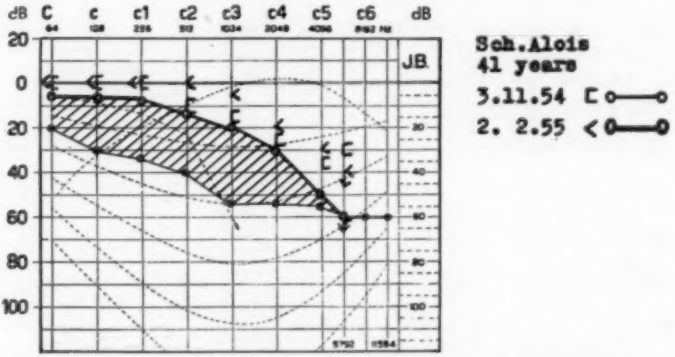

Fig. 11. 16.11.54. Tympanoplasty Type II with plastic-incus. Cholesteatoma, large marginal perforation and pulsating suppuration. Bony bridge removed. Head of malleus, incus and head of stapes defect. Both niches dissected from granulations. Handle of the malleus preserved with tendon of tensor tympani muscle. Palavit-staff under neck of the malleus to neck of the stapes. Implantation of amnion. Overlapping skin-graft. 2.2.55 New tympanic membrane mobile.

It can be seen that a high percentage of these patients have reached the 30 db line, theoretically for the Types I to III a satisfactory hearing ability; for the Types IV and V a good hearing ability. In the Types IV and V we even find a considerable number of patients in whom, without the apparatus of sound pressure transformation, results have been achieved, which in Types I to III would also have been called good. Since, for instance, in Type IV, the stapedial foot-plate lies open toward the auditory meatus and has not been touched, the good result can be due only to an increase in the sound

protection for the round window. In a small middle ear, built from the tubal corner, hypotympanum and round window niche, it is evidently quite often possible to increase the sound protection as compared with that provided by the normal drum if a dense scar is formed in the transplant which closes the reconstructed cavity. Still further increase in sound pressure difference between scala vestibuli and scala tympany may be obtained by sound reflection in the cavity to the fenestra ovalis or nor-ovalis. If in comparison to the "good" result in the last two types, the number of the "good" results in Types I to III is relatively small, we must take into con-

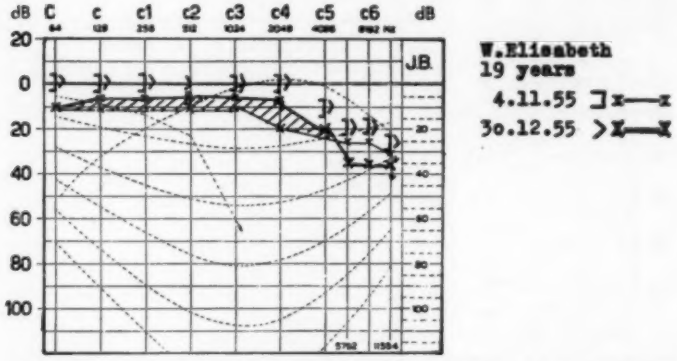

Fig.12. 10.11.55. Tympanoplasty Type III. Defect of the membrana flaccida, pars tensa preserved, cholesteatoma in the attic, matrix enclosing the ossicles. Bony bridge, incus and head of the malleus removed. Free skin-graft.

sideration that in many instances, especially in Type I, the air conduction curve, after healing, almost reaches the bone conduction curve, and only a conductive component of about 5 db will be left, which does not happen in Types IV and V. We must also consider that although the possibility of restoration of the sound pressure transformation exists in most of the tympanoplasties, defects of the ossicula are always signs of a long lasting granulating disease of the mucosa on the promontory and on the inner surface of the tympanic membrane. In this way the chances for the highest functional quality of the new middle ear are reduced.

In what manner do we have to plan and carry out the operation if we want to do it according to these principles? It is the first law in a tympanoplasty, like all other audiological operations, to remove no element of the structure of the middle ear before the whole middle ear has been examined with the operating microscope and the general plan of the operation, namely the type of the tympanoplasty, has been established. Frequently it is impossible to determine the type of operation before one has actually started. Remnants of the tympanic membrane, adhesions and granulations prevent us

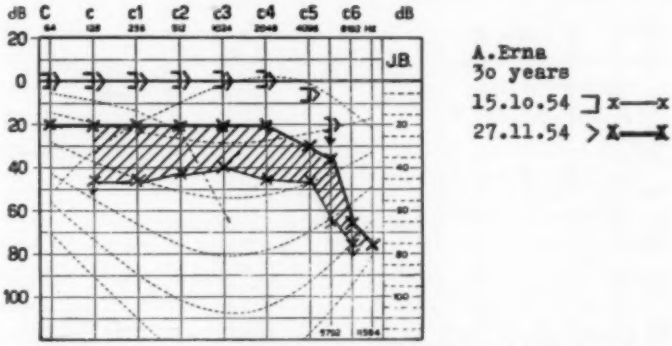

Fig. 13. 2-11.54. Tympanoplasty Type IV. Large cholesteatoma, total perforation, fetid suppuration, defect of the head of malleus of the incus and of the crura stapedis footplate covered with epidermis. Large fistula on the lateral semi-circular canal. Round niche dissected out of granulations. Free skin-graft. 27.11.65. New tympanic membrane mobile.

from reviewing the whole mesotympanum through the auditory meatus, and also prevent us from determining the acoustic functional ability of the two windows. The opening of the tube is often closed and the patency of the tube, therefore, not demonstrable. The severity of the infection of the mucous membrane might show considerable variations; for instance, there might be heavily granulating mucosa in the hypo and mesotympanum, whereas the antral mucosa might be almost without irritation or vice versa. For the microscopic examination of the tympanic and retrotympanic cavities without even the most minimal interference with the functional struc-

tures of the middle ear I have established three control entrances:

1. Control of the antrum.
2. The upper control of the middle ear.
3. The lower control of the middle ear.

For the control of the antrum I enlarge the bony auditory meatus a little bit backward and then open the antrum through a drill hole only closely behind a broad bridge which gives protection to the whole epitympanum and the ossicles. From

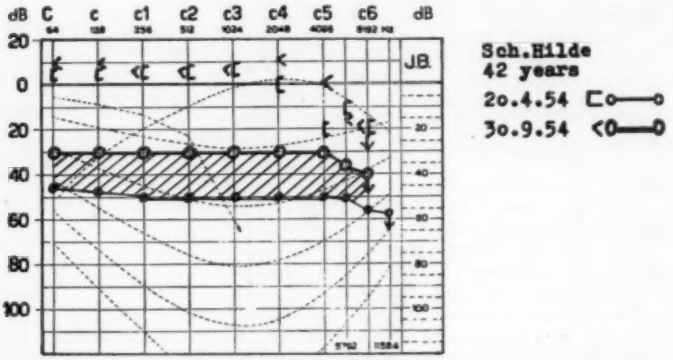

Fig. 14. Otosclerosis and central perforations of the tympanic membrane. 27.4.54. Tympanoplasty Type V. Small cholesteatoma in the attic, bony bridge preserved, fenestra in the semi-circular canal prepared. Free skin-graft. 17.9.54. Fenestration. Brother with success fenestrated for otosclerosis only.

here the pathological process in the antrum and also in the aditus ad antrum can be visualized.

The upper control of the middle ear is done through the removal of Shrapnel's membrane, which seals off the epitympanum but does not participate in the transformation of the sound pressure. From here control of the incus and malleus can be accomplished, especially the control of the long process of the incus, which is easily destroyed by granulations from the oval window niche and from the epitympanum. The

operation can, therefore, be shortened if one examines at an early stage this weakest point on the ossicular chain. The stapes and oval window niche can also be reached from here. After removal of a small marginal portion of the bridge with a diamond burr, one can satisfactorily visualize the epitympanum and a small sac-like cholesteatoma in Prussak's space can be easily enucleated.

In a larger cholesteatoma where the matrix is not yet attached to the whole attic and does not cover the ossicula but still can be removed as a sac *in toto*, I cut through the bridge with the finishing burr either below the tegmen tympani or closely above the facial canal, depending upon the place where the cholesteatoma sac extends into the depth. In this manner the ossicula can be preserved under the protection of a large part of the bridge.

The lower control of the middle ear is known through my method of fenestration, because I never create a new window before I have microscopically controlled, with preservation of the whole middle ear, the tympanic cavity; and especially both labyrinthine windows. Close to the floor of the auditory meatus, between annulus osseus and the lateral semicircular canal, more precisely, between chorda tympani and nervus facialis below the tip of the short process of the incus, an opening into the sinus tympanic is established with the diamond burr which will lead in a typical manner directly to the stapedial tendon. After the enlargement of the opening, the oval niche and the hypotympanum to the round niche will come into view. On still further enlargement toward the floor of the auditory meatus even the membrane of the round window can be identified on a view from behind and below.

Entrance to the oval niche can be gained from this point to such an extent that with this approach the direct mobilization of the stapedial foot-plate, especially around the base of the anterior crus, can be carried out. In this manner we are able to prove the function of both windows, before the incus is removed and possibly the crura of the stapes are infractured. It is furthermore possible to smooth out, with a fine diamond burr, a round window niche which had been blocked

with connective tissue. Thereafter an implant serving as substitute for the mucosa can be laid directly over the round window membrane.

In large perforations of the drum with both window niches exposed to the direct view, not all these controls are, of course, necessary. If, however, the posterior part of the drum over both windows is preserved and contains a good layer of collagenous fiber, it is in my opinion not advisable to lift it out of the annulus. Through the perforation it has already lost some of its stiffness, and in this way it might suffer a further loss of its functional quality.

I think that these operative controls are even then necessary when the chronic otitis shows findings which are most favorable for a tympanoplasty such as: central perforation; pars tensa well preserved; no discharge; mucous membrane of the middle ear, as far as visible, not irritated; good increase in the hearing ability even in the threshold audiogram by closure of the perforation through an artificial drum. The marked increase in hearing with an artificial drum is often not successful at the first attempt; but if we are successful at least once, it is evident that both labyrinthine windows are able to function properly. The lack of an increase in hearing after repeated attempts does not, however, prove that there is a fixation of one of the two windows; there could be a blockage of one of the window niches, or it could have different other causes. Even under the most favorable conditions of a central perforation one often finds on control of the sinus tympani, the round window niche or the epitympanum loose granulations which completely fill up these narrow spaces. I have observed young patients for many years, before the operation, who had first a very good hearing gain on closure of a perforation which later on decreased. On operation a progressive scarring of such granulations could be seen. Through an operation which solely closes the perforation (myringoplasty) one can easily get a good initial result; but there is great danger that after several years, with progressive scarring, the hearing ability will go down below the preoperative level. A revision of the first operation is less likely to suc-

ceed, because extensive masses of scar tissue must be removed and large areas of the bony wall will be without a mucosal lining. Myringoplasty with Thiersch grafts were tried 70 years ago by Berthold and Tangemann, but were abandoned.

The results in the different types of cases indicate that the theoretical considerations regarding the audiology of these operations are correct. We shall now see a sample of each of these different forms (see Figs. 11, 12, 13, 14).

The theory and practice of these operations which show a great variation I could only briefly demonstrate. They can only be done under the microscope and they are very strenuous and time consuming; but all this is worthwhile, because a definite healing of the chronic otitis media and a large percentage of good functional results can be achieved. I believe that all audiological operations are interrelated: fenestration, mobilization and tympanoplasty in chronic otitis media, and in adhesive changes after middle ear infection; malformations of the middle ear and atresia of the external auditory meatus belong to the same group of cases. On this occasion the operation for the middle ear follows the same principles as the tympanoplasty; with a movable stapes a columella-effect might be advisable and with ankylosed stapes, a fenestration.

SIXTH INTERNATIONAL CONGRESS ON OTOLARYNGOLOGY.

The deadline for submitting titles of papers and films for presentation at the Sixth International Congress of Otolaryngology is Oct. 1, 1956. Requests for places on the scientific program must be received before that date together with an 800-word abstract of the paper. Applications and abstracts should be sent air mail to the General Secretary, 700 N. Michigan Ave., Chicago 11, Ill., U. S. A.

"SICK PEOPLE IN A TROUBLED WORLD."

HOWARD A. RUSK, M.D.,*† (By Invitation)

New York, N. Y.

I would like to first discuss briefly rehabilitation in general and then talk about some of our mutual interests. Rehabilitation has really become a $64,000 word. It means so many things today that it means very little. It may be used in discussing a program for the economic recovery of a country, a business; it may mean the bringing back to optimum of a severely disabled individual. In our own department we call rehabilitation the third phase of medical care, the first phase obviously being prevention; the second definitive medical and surgical care, and the third phase is that which happens to the individual after the stitches are out and the fever is down.

I would like to outline for you briefly the problem that you and all of us in the health services have had a great part in creating. Along with improved knowledge of nutrition and a better standard of living, we have raised the age expectancy in this country from 46 at the turn of the century to, as of last month, the Biblical three score and ten for the first time in the history of the world. This has created a problem; it means that today in the United States we have 14,250,000 beyond the age of 65. By 1980 with our present medical knowledge alone they will exceed 20,000,000. Of this 14,000,000 or more, one-third have no income at all. One-third have less than $500 a year, and of the remaining one-third, 50 per cent are self-employed. A man today of 65 has a life expectancy of just over 15 years; a woman 17.6 years. This has created a special problem best summarized in a sentence by George Morris Piersol some years ago, when he said, "We have added years to life; it is also our responsibility to add life to years." The first problem, therefore, is that of retirement.

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* Presented at the Sixtieth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Montreal, Canada, May 15, 1956.

† A verbatim transcript of an extemporaneous address.

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Everyone knows that age is physiological and not chronological, that some are old at 50 and others are young at 80. By the willy-nilly employment of the retirement practice that is currently in vogue, I feel that we are wasting the most precious human resource that we have. That resource is wisdom! People are not born wise; you may be born brilliant, but wisdom comes only with experience and experience comes only with time.

I wonder how world history would have been written had this retirement policy been adhered to. It would have meant that Winston Churchill would have retired a year before the Battle of Britain; that Mr. Baruch would have retired 21 years ago, before the rubber program and before the atomic energy program, and we would have lost all of those years of his wise counsel.

I, for one, feel that because we in medicine have taken the leadership in prolonging life, we also must provide leadership in meeting this problem; and I think that any country which can solve the mysteries of electronics, radar and atomic fission can certainly put the pieces together to know who should continue to work without detriment to his own health or danger to his fellows. Industry is certainly able to know who can continue to work and be valuable as a producer. This is the No. 1 problem that we face.

Second, we have all of the problems of disabilities that come with an aging population. We became tremendously disturbed in this country about the problems of infectious disease and have done something about it.

In an epidemic of polio we have, I think, approximately 52,000 cases. Today in America, not including Canada, we have 1,500,000 individuals who have had strokes of apoplexy. In the past they were felt to be hopeless; they are not hopeless at all. Winston Churchill again is a good example.

We just finished an anlysis of our first 1000 at Bellevue. The average age was 63. The average time from stroke to beginning training, nine months. The average training time seven weeks; 900 of the 1,000 we were able to train to the

point where they could meet all the needs of daily living and ambulate and live non-institutional lives, and 400 went back to some kind of gainful work.

In this third phase of medicine every time we make medical advancement we compound the felony, as far as we in rehabilitation are concerned, because people stay older longer to get all the disabilities that come with age. I think, too, that this realization brings a new and very basic concept which, I believe, that we in medicine have failed to remember, just as the general population has failed to remember. Not for hundreds of years has society paid for strength. It pays only for two things: the skill in your hands and what you have in your head; but if you are honest and let your subconscious reflection prevail, physical perfection and ability will seem synonymous.

The average person uses only 25 per cent of his physical capacity in daily living, but the reason we can do a pretty good job in the practice of medicine is because nature has given mankind such terrific powers of healing and of physical compensation. The blind man sees with his sense of touch and with his sensitized hearing. Put that man in a job where he can use those overdeveloped senses, and every experiment has shown that he will outproduce the normal man from 20 to 30 per cent. Put the paraplegics at a bench job where they can use the hypertrophied muscles in the upper arms and shoulders, and they will kill the ordinary worker doing the same job and outproduce him every day in the week, because they are working with hypertrophied muscles.

I will give you an illustration of just how easy it is to forget. About four years ago I was at a meeting in a western city and had a call from a young woman physician. She asked whether she could see me. "Certainly! Would you like to come by the hotel?"

"I am a paraplegic in the hospital," she said. "It is impossible, but you are speaking at the hospital tonight. Can I see you after the meeting?"

I said, "Certainly."

"You will recognize me. I am a paraplegic. I am in a wheel chair."

Her story was this: She was 32 years old, had five years training in medicine, neurology, psychiatry at Bellevue, had gone back to her home city, was instructor in medical school, and was assistant to the leading psychiatrist in this city. Fourteen months previously a psychotic policeman had come

With advice of work simplification and home management, many women, despite severe disabilities, can assume their roles as homemakers.

into her office looking for the boss. Not finding him there he had pulled out a gun and shot this girl five times. One bullet transected the cord at T-10. The intestines were punctured in dozens of places; also a bullet wound through the liver and one in the right thorax. She had magnificent treatment; otherwise she would not have survived. The internist took care of the antibiotics, the blood, the electrolytes and the fluid; and the general surgeon sewed up the rents in her intestinal tract

and stopped the bleeding in the liver. The neurosurgeon did a laminectomy. The surgeon stopped the bleeding in the chest. This was 14 months ago, and this was the first night she had worn a dress.

I said, "What is your treatment?"

"Massage to the lower extremities 20 minutes three times a week."

I said, "Are you practicing medicine?"

She looked at me like I was crazy. "My surgeon said to me, 'let's face it; you are all washed up as far as medicine is concerned.' One day one of my colleagues came by and said, 'Why don't you come down to the clinic on my day? You are perfectly capable of filling out X-ray requisitions.'"

I said, "I've learned something. I always thought you practiced medicine with a trained mind, dedicated heart, eyes, ears, your hands, personality and your voice. All of those things you have. I didn't know that feet were a necessity."

She smiled wanly and said, "I don't know what I am going to do. I have a $70 month pension, a two-year-old baby; my husband has left me and I owe $3,000 for a hospital bill." I said, "Certainly something can and will be done."

I went home the next day and worried about this girl all weekend, and Monday afternoon called her and asked her when she was coming to New York to take her new job.

She said, "What job?"

"You have just been given a two-year appointment as assistant resident on the rehabilitation service."

She said, "When do you want me? Mind you, I have never been out of the hospital."

I said, "When convenient."

In 48 hours she arrived. We did not put her in the hospital. We put her in the residents' quarters, and placed her in the training program, training herself half a day and seeing patients half a day. That was the 25th of March, 1951.

On Labor Day she walked on her braces and crutches to a taxi, went to the airport, walked up the ramp, flew back, and spent two weeks with her child. She finished training, qualified for the Boards, took her examination and finished in the upper 10 per cent, and that young woman now is the Acting Chief of the Rehabilitation Service on the Neurological Service in the National Institutes of Health. At home in a great medical center, one of her own faculty forgot what was needed in the practice of medicine. Now that girl is a better doctor in this field than any whole person can ever be, because she will teach both by precept and by example.

What is this rehabilitation service? What do you do? It is very simple. Our concept is that we are a service department like the laboratory and the X-ray, and in the definitive stage of medical care, we can act in a consultant capacity.

At Bellevue, for example, we see all elective amputations before the member is amputated. We tell the individual why he cannot keep the stump on the pillow, and the exercise he needs to prevent the flexion contractures. We tell him what an artificial limb is, how much it costs and how long it takes to get it; that he has to be trained to use it, and often we will bring an amputee with us to demonstrate. In the older age group, with poor balance and a problem in the other leg, or a cardiac problem, we will begin to discourage him on a prosthesis even before he is operated upon, and tell him what he can do in a wheel chair.

When training becomes primary and definitive care secondary, then we like to accept primary responsibility. Patients come to us; we are a professional service and not a technical service. We reserve the right, as you do, to examine the patients referred to you and either agree or disagree with the diagnosis, and that we do.

To show you the need of this particular phase I will cite briefly a case that came to us two years ago. A young man who had been in a terrific automobile accident with severe brain injury, unconscious nine months, tube fed, three craniotomies, contusions of the brain, and when he finally woke up he had a severe cerebral ataxia and a terrific speech defect.

You know the brain syndrome and how difficult and discouraging it is to treat, probably one of the most difficult that we see in adults. He finally got on his feet. He was sent as an ambulatory patient to a training center in the city in which he lived; 12-pound leads were put on each shoe and wrist leads to try to decrease the ataxic movements. He went into speech therapy, but his mother complained bitterly that this boy

Such simple things as getting from the wheel-chair to the bed are the foundations of self-sufficiency for the severely disabled.

wasn't getting along in speech, and the reason he could not speak was that he was dyspneic, and something had to be done. They were told over and over again that this was a severe injury, that it took a long time and that they must not be impatient.

When the Fellow on our rehabilitation service did the original examination, he was interested to note that this boy had a dextrocardia. He was also interested to note there were more

intestinal than breath sounds in the left thoracic area, and a barium swallow showed about two-thirds of the intestinal tract in the left pleural cavity. Walking with 12-pound leads is not specific for diaphragmatic hernia.

He was sent back to the surgeon, who closed the rent, and strangely enough the boy could speak a great deal better. This is the first phase of treatment; then we do a careful muscle study; range of motion and definitive muscle examination, but the heart of our program is simple. We test for 137 inherent needs in daily living, things that you do every day subconsciously. The following questions are asked: Can you brush your teeth; can you comb your hair; can you feed yourself; can you dress yourself; can you turn from side to side in bed; can you put on your own braces; can you get from bed to wheel chair; from wheel chair to toilet; from wheel chair to bath tub; from bath tub to wheel chair? If you walk, can you step up and down the curb? (If you cannot, walking does you very little good.)

About five years ago, Dr. George G. Deaver, my associate at New York University, reviewed 500 cases of polio, 16 years or older, which had the disease five years or longer and were confined to their homes, to ascertain why. He found 70 that with adequate training would not need to be thus confined.

The first girl we brought in was 28, had polio when two. She was a quadriparetic, she had had 26 assorted surgical procedures, six spinal fusions, could walk only with a crab-like gait on crutches, and could not get up and down a curb. She had had a home teacher, learned to type, and could type 30 words a minute accurately. This did not help her, as she could not go out, nor could she get up and down the curb to cross the street. It took seven months to teach her a technique by which she could get up a curb by swinging the leg back, catching the heel and turning a certain way. I happened to be in the gym the day she made it the first time. The patient screamed like a cheering section of the football game. Once she learned the trick she did it a dozen times the first day, 25 the second and 50 the third. That was Friday; she went home on Monday and took a full time job at the switchboard at

Bellevue; that was five years ago, and she has never been late a morning; never missed a day. She waited 26 years, because there was an eight-inch wall around her house.

I tell the story because it exemplifies the necessity for attention to detail and this is the principal factor in the training program for renabilitation.

Wherever possible group instruction and training is given at the Institute of Physical Medicine and Rehabilitation.

Then our patients are psychologically screened, and have an interview with our vocational counsellor. We begin to talk about work as soon as we see the patient, and then the patient comes before the whole staff. The program is planned, and these individuals go back to school under a planned program, going to class five hours a day.

Hemiplegia is the most common disability we see. The average training time of hemiplegia is six weeks. The most difficult problem in training the hemiplegic which you see is aphasia. About 40 per cent of the patients we see

are aphasic. We have learned a number of simple things about the problem of aphasia and not too much about the long tedious training.

In the first place in this country we have fewer speech therapists than any of the other co-professional personnel. I should say that if every school that has a class of speech therapists doubled its class every year for the next ten years there would still be too few speech therapists.

The first thing that must be done for the aphasic is that he must be assured that he can be helped. We have seen patients who have been in the most absolute agony for two to four months because of aphasia. Nobody had taken the time to explain this simple thing.

It takes careful evaluation by a trained person to know the type. Unfortunately, most of those we see are mixed, and it takes continuous training over a considerable period of time. Let me suggest one thing in the treatment of aphasia: Do not be too positive in your prognosis, because when the edema subsides in the healing area about eight weeks after the original cerebral insult, the outlook and prognosis often change totally. The same is true for the diagnosis of organic brain damage in the hemiplegic patient, because until this first acute phase is over, you cannot tell how much the patient will be able to do.

Paraplegics have been a very interesting group with us, and we have now seen nearly a thousand. The program started in the services during the war. In World War I we had 400 paraplegics; they were no problem then, because one-third never got back to this country. Among those who did return another third did not survive 60 days. The mortality was more than 90 per cent in the first year. There are only two living today from World War I that I have been able to find, and they were not total, but partial.

In this last war from combat we had 2500. They were a real problem. This time knew about urology and nutrition and the management of decubitus ulcers and we had antibiotics. They did not die. They were young men who had given the

It is difficult if not impossible for the non-disabled to understand the emotional satisfaction which comes to the paraplegia the first time he stands again.

best of themselves to the country. They had strong arms and good minds. They wanted to live the best lives possible with what they had left.

I can say that the dynamic program in the VA hospitals is magnificent.

Out of the original 2500, 1763 today are living in their own homes, driving their own cars and 80 per cent of that group are either at school or in some kind of job.

It might interest you to know, however, that the case load in VA hospitals has not decreased by a single case. For every paraplegic who has gotten out, another from the casualties of civilian life has taken his place. This is a hard lesson to learn.

In the early days after the war we all thought about disability in terms of the veterans. The veterans are a drop in the bucket. The first ten days on the Normandy beachheads we lost 11,000 men killed and wounded. In the same ten days we had 26,000 civilians killed and disabled on the highways alone, and that was with gas rationing. The three days of the bloodiest fighting of the war were the three days at Iwo Jima. We have four times as many individuals killed or wounded by farm accidents alone each year in this country as were lost at Iwo Jima. I think the general figure now is ten to one.

The average training time for the clean paraplegic, T-10 or below, is 120 days. I say "clean" because it takes us twice as long to clear up the complications due to faulty early management, bed sores, kidney complications and contractions, all of which can be prevented, than it does to train the patient.

We have to change our thinking about the moving of these patients. We have seen nine patients in the last two years in whom we were perfectly sure that the cord was not transected by the accident but by the moving of the patient; because the textbook says that these patients should be moved in hyperextension. You do not know how these patients should be moved until you see the position of the fracture; therefore, they should be moved on a hard surface in a neutral position.

One of the most poignant stories I ever heard was one of a young Naval aviator who crashed the plane. He was able to crawl away while it burned. He had sensation and motion in his feet. He was picked up by the aid men who had read the book and who forced him into hyperextension. On the 15-minute ride to the hospital his legs went to sleep. When he arrived he was paralyzed; his cord had been transected en route.

The children's program is a particularly interesting one. Here, too, we have brought it on ourselves, because the children

who formerly would have died, do not die today. We just finished an interesting series of spina bifida, and I think that every one who remembers the pre-antibiotic days, recalls an experience with this tragedy of the obstetrical ward. When the spina bifida child was born, you had to tell the parents this horrible thing, and the child was kept in the hospital. You and I knew that soon the child would turn, break the sac, get excoriations and ascending meningitis, and we would work vigorously together. We remember the days when you put urethral catheters in the spinal canal and washed it out with mercurochrome and anything we could lay our hands on. None of the spinal bifida cases ever recovered, and we felt relieved, and so did the family; but they no longer die. They live as paraplegics, and more than 80 per cent of them have good minds and strong hands and feel that they have a right to live the best life they can with what they have left.

We have just run our first series of 50, all types, and 35 are back in school. More than 80 per cent have bladder and bowel automaticity. Once braced and on their feet, they start to grow.

These are little children who will make their places in the world, because their life will depend upon the capabilities of their hands and their heads. Although they cannot run so fast, in some ways they have an advantage over normal children.

We have as consultants all of the specialties in medicine and one of the most valuable is the otolaryngologist. We could not make definitive diagnoses of speech disorders without you. Every child that comes on our service has a screening test for hearing acuity. We have a boy in the institute now who has spent eight years in an institution for the feebleminded, when he was only deaf. Once he was properly fitted with a hearing aid and began to learn lip reading his retested IQ was just double that of the original. It was a tragedy that he had to spend eight years before this simple test was done.

We saw a patient recently who had had an ascending viral meningitis who was diagnosed as aphasic, because they did not have a good otolaryngologist to examine and find there

was because no otolaryngological examination had been made which would have disclosed a muscular paralysis. With simple muscular exercise and chewing gum, this woman is speaking audibly and quite well in less than 10 days. She learned to walk also, for she had a hemiplegia, and her total training time in the hospital was less than five weeks.

I believe that you have a problem in your profession, just as we have in general medicine. They say as far as those of us in internal medicine are concerned, between 80 and 90 per cent of all the patients that come to the internist's office are not problems of organic disease, certainly not acute infectious disease, but problems in the chronic disease field or in the psychosomatic field.

I say to our freshman class every year, if in all the time you spend with us you get only one thing, I will say your time has been well spent, and that one thing is this: If you can get the same inner satisfaction out of taking an old hemiplegic out of a wet bed, teach him to walk, dress and feed himself, talk, be dry, and to live outside the hospital and to get something out of his life if you can feel the same way inside as you now feel out of making a diagnosis of histoplasmosis or Kala-Azar, which you may or may not see in a medical lifetime, then your life is well spent, because these are the patients that you are going to see in practice.

If you do not know how to manage a hemiplegic, you will be a "barbital-potassium iodide" doctor. I speak feelingly because I was one for a long time; 17 years in internal medicine. I knew how insecure I felt when my hemiplegics came in. I had a technique. I put the blood pressure cuff on. They wanted to talk. I did not have time to talk. I had to take the blood pressure. If they talked I did not hear them. Before they had a chance to talk I would say "You are doing fine," and I would give them a mild sedative and hope they did not come back for a long time because I felt insecure.

I believe that you have the the same situation, in that your problems have changed with the antibiotics, and the young men coming along in your field are going to have to get the same inner satisfaction out of caring for the cerebral palsy child who has a problem in speech and hearing and also the

Ninety per cent of all hemiplegia patients can be retrained to perform all of the activities of daily living.

old people. If young men can feel the same about their problems as they did in their younger days about a fresh, acute mastoid as a life saving, dramatic procedure then their professional experience is going to be richer and more profitable to their patients. These are the kind of patients that they are going to see.

I am frequently asked how much of rehabilitation is physical and how much is emotional? I always answer the question the same way. If you do a day's work, how much do you do with your right hand and how much with your left? In the

first place, it depends upon whether you are right or left handed; second, it depends upon what kind of a job you have. If you know both of those factors at the end of the day you cannot tell. It is total.

We did a three-year follow-up on 146 patients who had been through our program in the early days, and we found that 55 per cent of them had had real emotional problems. Some of these problems we met, and some we failed to meet. I think we are meeting practically all of them now. It is silly to teach these persons to crutch-walk if they are not to go out of doors. The push has to be the satisfaction. It has to be the vocational, medical, physical, emotional; total.

We have here not only a medical problem but also an economic problem. If we do not do something about meeting this problem, by 1980, for every ablebodied worker in the United States there will be one individual beyond the age of 65, one chronically ill or one physically disabled worker, in addition to his dependent family. The economists tell us that the present situation is economically unfeasible.

We have a special responsibility in medicine and a special opportunity as well, for we have a tool in medicine, and especially in the programs for disabled people, that can build a firm foundation for international understanding. If we do not have that we cannot have peace.

My interest in this program began in 1949 during a mission to Poland. I went three weeks after Mr. Field disappeared at the airport, a very disconcerting time. I was sent by the United Nations as a consultant to help Poland set up a rehabilitation program. Poland was on her knees then; Warsaw was more rubble than city; the people were hungry, cold and bitter, and in a desperate situation.

When I left I thanked the Minister of Labor but he said, "Thank *you* for this mission. This has been a good mission. Not only have we learned something to help us with our disabled, but more than that we have learned that Poland and the United States have many common problems." He said, "You know, if we could just work on these together, one day

maybe we could understand each other well enough to live in peace together in the world. That has to be our objective."

I said, "Those words were worth the mission to me; will you put them into writing?"

He said, "You will have them before you leave." As the train pulled out of this rubble pile, which was a station, a man in a greenish black suit stepped out from behind the rubble and handed me an envelope.

The doctor who took me about on that mission was the professor of Orthopedics of the University of Posnan, a distinguished physician. I remember the first morning that we went into his study. It was cold; they turn the heat on there by the calendar, not by the temperature, and we had a cup of tea. There was a magnificent portrait of a young physician on the wall, black hair, long white coat, stethoscope in his pocket. I said, "Prof. Dega, that is a wonderful looking young doctor. I hope I get to meet him."

He said, " You won't meet him; he is dead. He was in Warsaw during the uprising, and there was a child in the ghetto who got acute osteomyelitis in the late afternoon. Word came to this young physician through the underground. He knew that if the child was not operated upon that night, it would not survive. He went into the ghetto, and in the midst of the operation the Nazis came with machine guns and killed him, the patient, and everyone in the operating room."

Prof. Dega came to two meetings recently, the International Poliomyelitis Congress in Rome and International Society for the Welfare of the Cripples' Meeting in The Hague. Day before yesterday I received an official request from the Government of Poland applying for membership in the International Society for the Welfare of Cripples.

I have seen this happen in Israel, Greece, Korea, and all over the world in medicine. We have a common language. Osler said it simply in his early days of writing: "Medicine knows no nor has it ever known an international boundary." If a man's legs are off or if he is deaf or blind, the basic problem is the same regardless of his religion, politics, color, geographic

Good fitting and sound training in the use of prostheses are as important as the prosthesis itself.

origin." I have never found any place in the world that you could not talk about these problems on common ground.

I feel that we have not done enough throughout the world in this program. We have spent millions of dollars in our aid programs, and I am afraid that sometimes we have forgotten people.

I ran across a little note in a medical journal last week that put this in high focus to me. A little hospital medical society in Great Britain sent to the medical school in Afghanistan four boxes of pathological slides. They were so excited about

this gift coming that they had a reception committee to meet the airplane to take the box of slides from the Air Express.

Six weeks ago Alfred Vanderbilt, who is deeply interested in the World Veterans Federation, was in Djakarta, Indonesia. He cabled me, "Could you send me a modern artificial arm? No one out here has ever seen one."

We made one. We did it in plastic, in the color of an Indonesian's skin. It was sent Air Express. The government officials met the airplane to take the arm. It was passed from hand to hand, and finally fitted on an Indonesian whose stump it fitted. Alfred Vanderbilt said it was the high point of the meeting.

In the U. S. we have spent a million dollars a year for ten years in a prosthetic research program for veterans. They have the finest prosthetic devices in the world today. Now we are beginning to share them with civilians and starting on a children's program. I say we should share them with the world. I think the sharing of our prostheses would probably bring more understanding in the backward countries than the sharing of peace time atomic energy, because this is something people can see and feel. If you have been hungry and then well fed over a long period of time you forget how you felt when you were hungry. If you had had malaria every summer and then not had it for a decade you forget what the chills are like. If you see someone crawling on his hands and knees and who then walks again, and you know who taught him to walk, it is a different story. You do not forget.

So it is my deep hope that we can use our tools for a program of international understanding, and feel that we in medicine will take our responsibility as well as the great strength; we have this tool to add not only to medical knowledge but to world understanding as well.

A British philosopher said it 100 years ago so simply: "If every man would mend a man, then all the world be mended."

My gratitude to you all.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The meeting dates of the Sixth International Congress of Otolaryngology are again emphasized as May 5th through May 10th, 1957. The scientific program for the Plenary Sessions is now complete and is as follows:

CHRONIC SUPPURATION OF THE TEMPORAL BONE.

OPENERS: Marcus Diamant, Central County Hospital, Halmstad, Sweden—Anatomical Etiological Factors in Chronic Middle Ear Discharge.

Luzius Rüedi, Zurich, Switzerland—Pathogenesis and Treatment of Cholesteatoma in Chronic Suppuration of the Temporal Bone.

Horst Wullstein, Director, Otolaryngological Clinic, University of Würzburg, Germany—Surgical Repair for Improvement of Hearing in Chronic Otitis Media.

DISCUSSERS: A. Tumarkin, Liverpool, England; Juan Manuel Tato, Buenos Aires, Argentina; T. E. Cawthorne, London, England; Fritz Zöllner, Freiburg, Germany.

COLLAGEN DISORDERS OF THE RESPIRATORY TRACT.

OPENERS: Hans Selye, Director, Institute of Experimental Medicine and Surgery, University of Montreal, Faculty of Medicine, Montreal, Canada.

Introduction:

Michele Arslan, Padua, Italy—The Upper Respiratory Tract.

Leslie Gay, Physician-in-Charge, Allergy Clinic, The Johns Hopkins Hospital, Baltimore, U. S. A.—The Lower Respiratory Tract.

DISCUSSERS: Victor E. Negus, London, England; Branimir Gusic, Zagreb, Yugoslavia; Aubrey G. Rawlins, San Francisco, U. S. A.; Henry L. Williams, Rochester, Minn., U. S. A.

PAPILLOMA OF THE LARYNX.

OPENERS: Jo Ono, Tokyo, Japan—Etiology.

Plinio de Mattos Barretto, Faculty of Medicine, University of Sao Paulo, Brazil.

Diagnosis:

F. C. W. Capps, London, England—Therapy.

DISCUSSERS: C. A. Hamberger, Göteborg, Sweden; Pedro Hernandez Gonzalo, Havana, Cuba; Eelco Huizinga, Groningen, Netherlands; Albert von Riccabona, Vienna, Austria.

Applications to present voluntary papers are being received regularly and anyone wishing to present such a paper should make known his intentions prior to the deadline of Oct. 1, 1956.

The motion picture sessions, as well as the scientific exhibits, will prove to be outstanding in every respect. It is essential that anyone wishing to present an exhibit should make his intentions known immediately, as the final date for consideration is Aug. 1, 1956; applications to present motion picture films should be sent in before Oct. 1, 1956. Anyone planning to attend the Congress and who has not yet registered should do so immediately in order to obtain hotel registration priority.

For more detailed information pertaining to the Sixth International Congress please communicate with the General Secretary, 700 N. Michigan Ave., Chicago 11, Ill., U. S. A.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. John R. Lindsay, 950 East 59th Street, Chicago 37, Ill.
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Secretary-Treasurer: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Statler Hotel, Washington, D. C., May 4, 1957.

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Meeting: Statler Hotel, Washington, D. C., May 3, 1957.

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Meeting: Sixth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Brazil, 1958 or 1959.

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Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles, California.
Time: 6:00 P.M., first Monday of each month from September to June inclusive—Otolaryngology Section. 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section.

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